THE Journal Kansas Medical Society

AUGUST
1955

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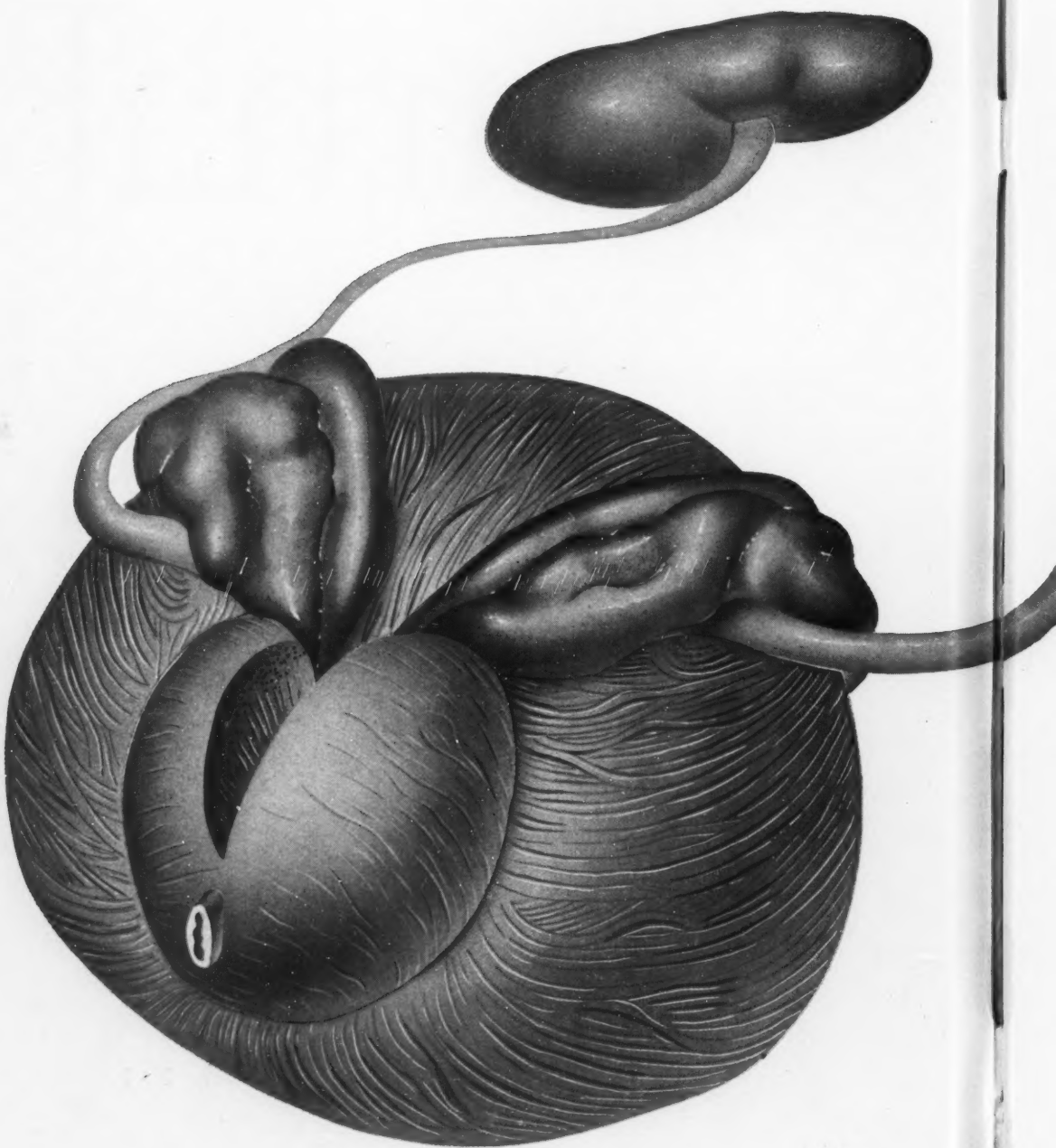

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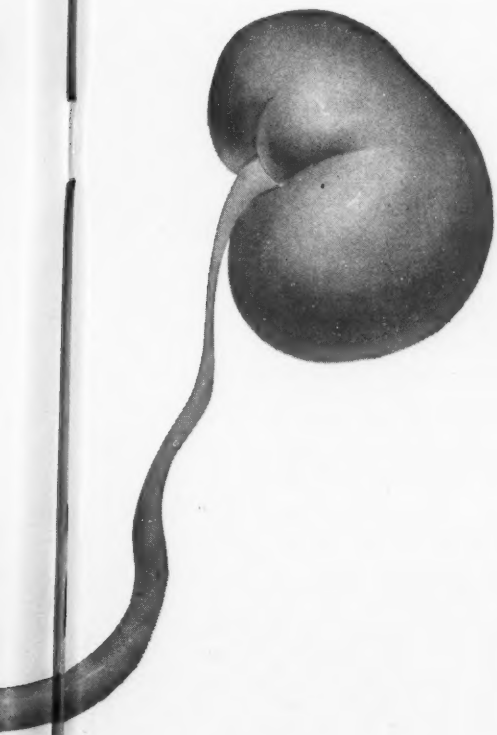

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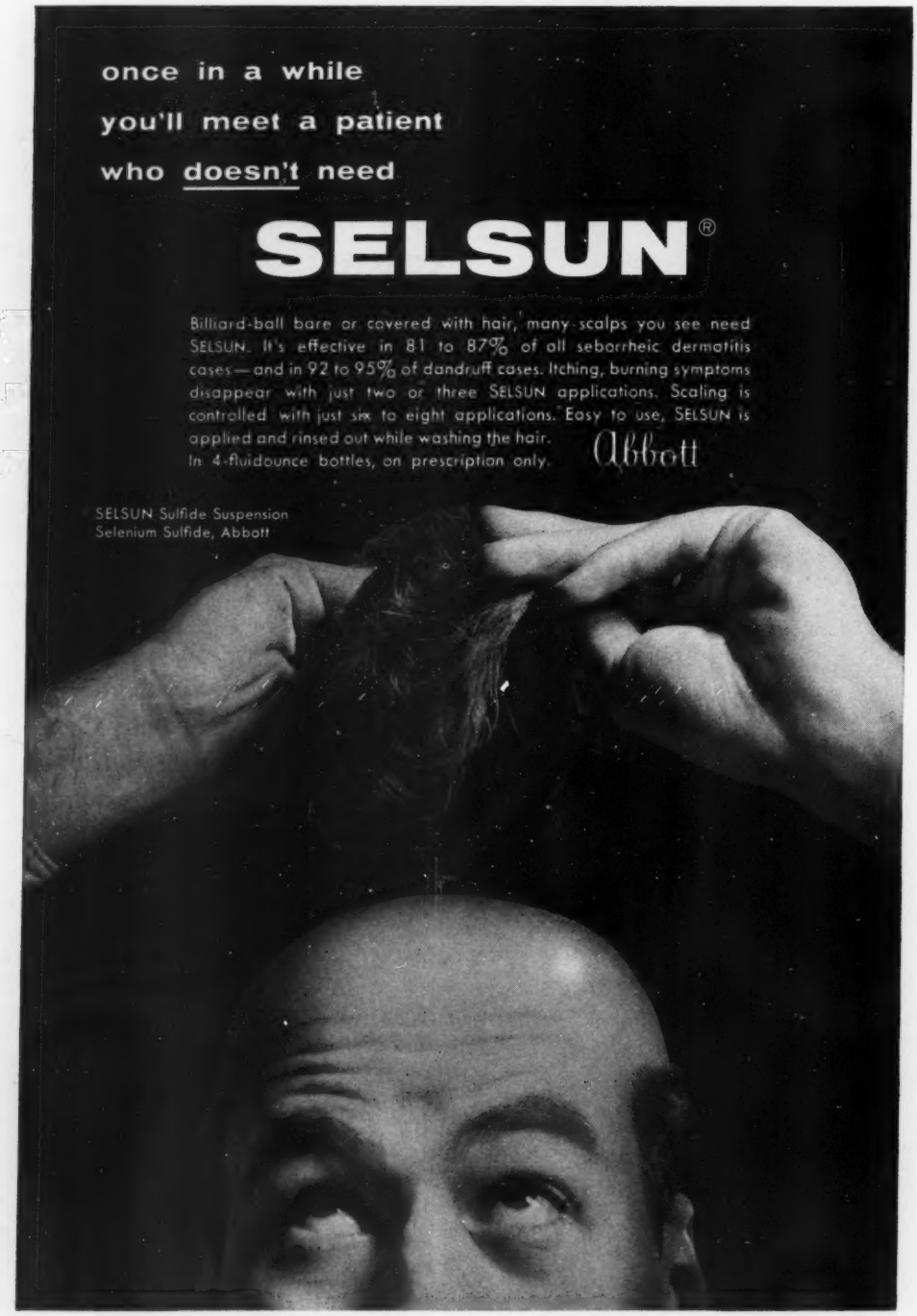
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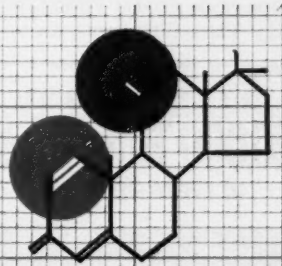

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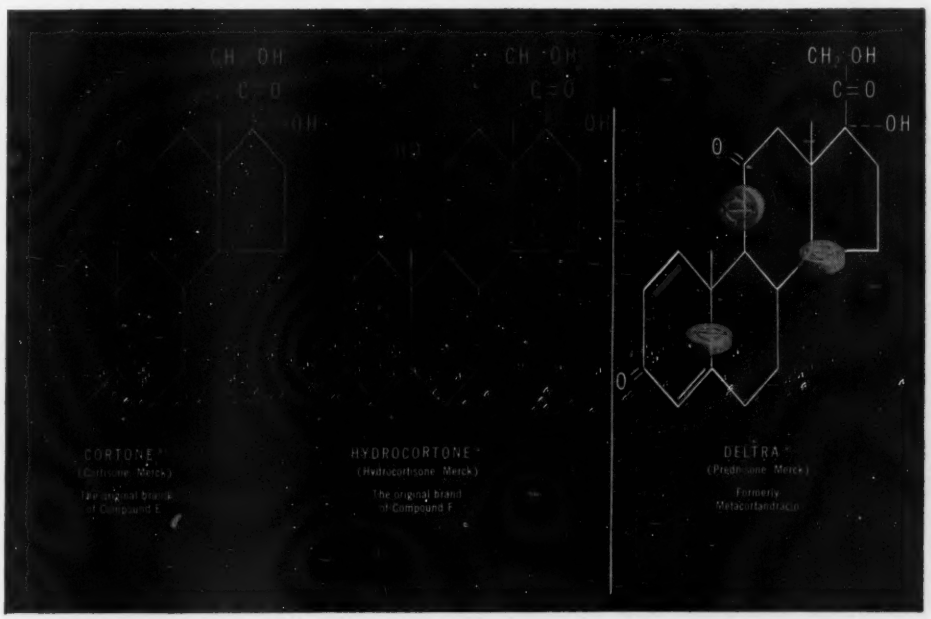

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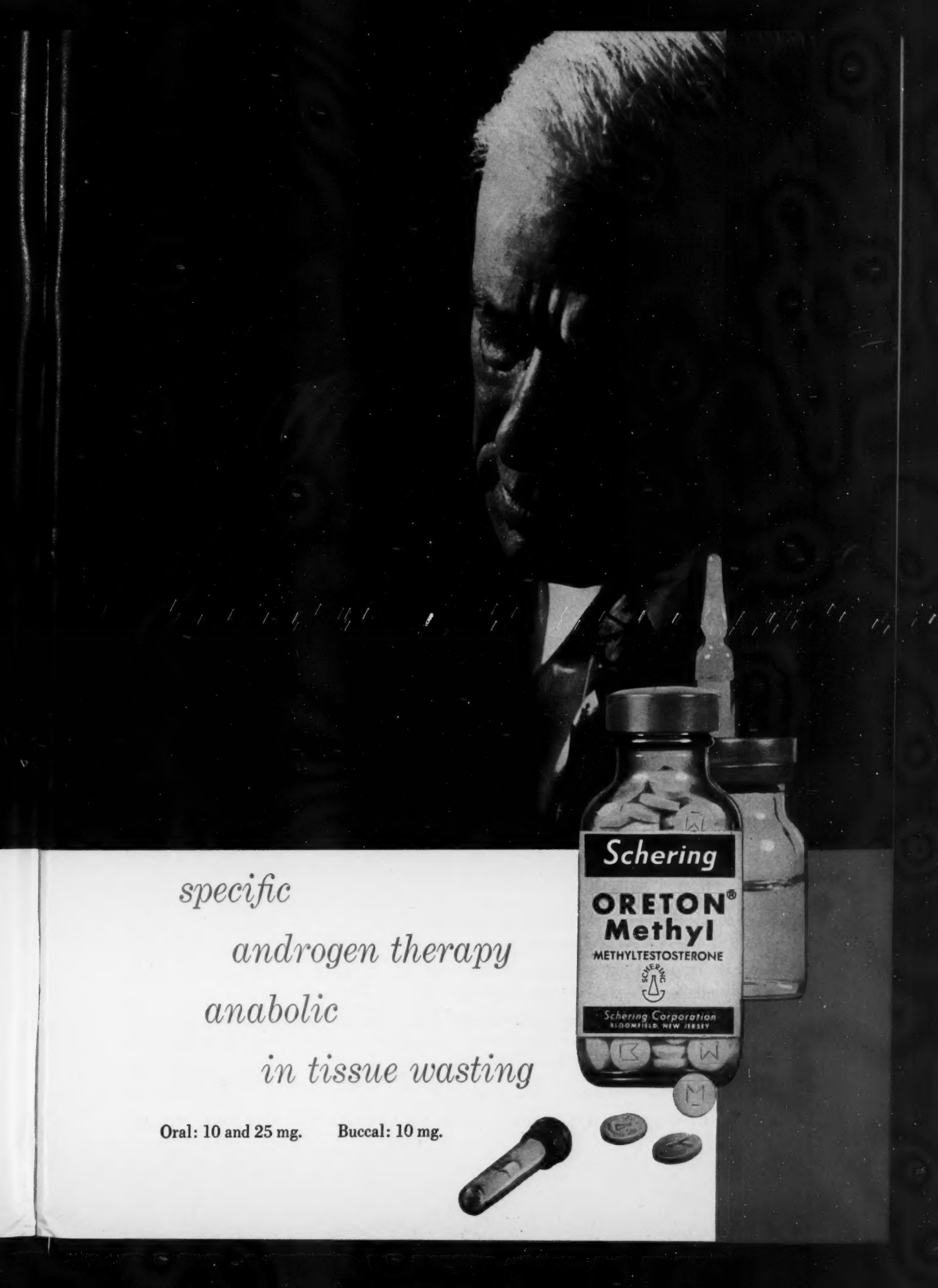

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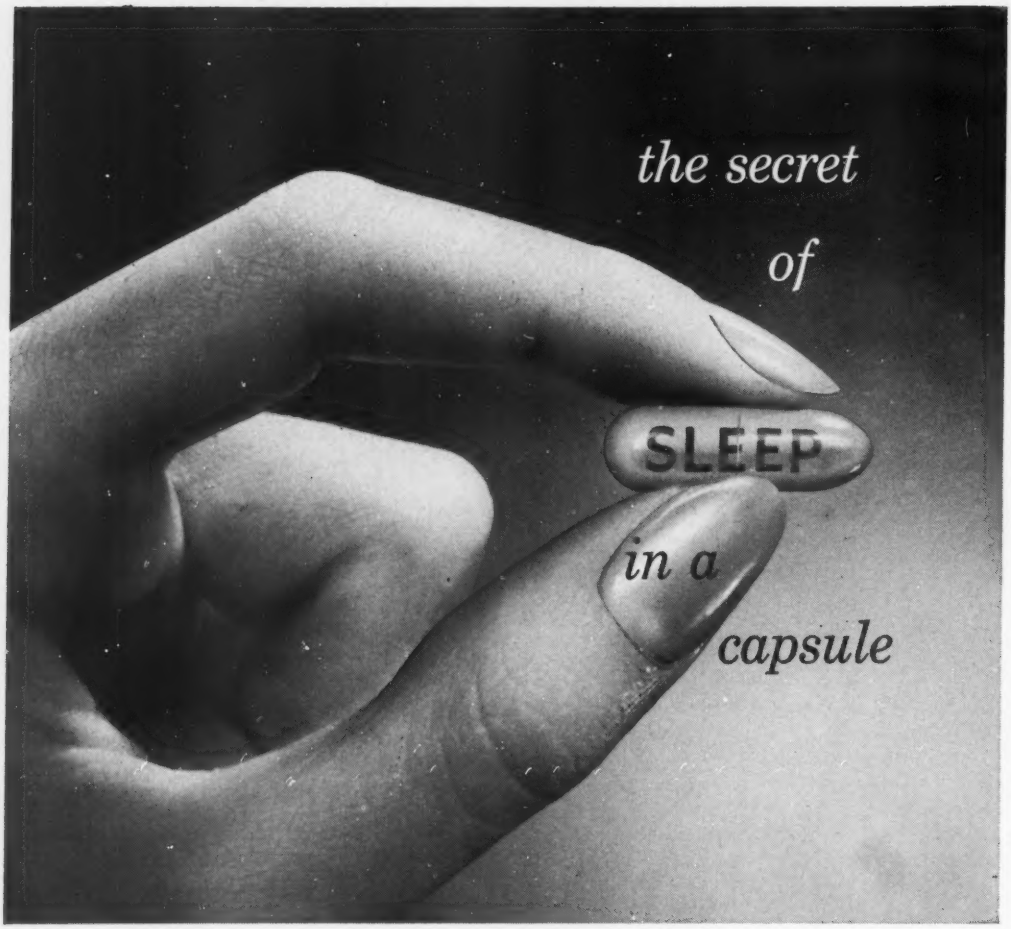

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Volume LVI AUGUST, 1955 No. 8

The Surgical Treatment of Gastritis

John G. Shellito, M.D., and B. E. Stofer, M.D.
Wichita, Kansas

One of the most controversial subjects today is that of chronic gastritis and its relationship to carcinoma. Konjetzny[15, 16] was one of the first to associate carcinoma of the stomach with chronic hypertrophic gastritis. Hurst[12] in 1929 reviewed carcinoma of the stomach and gastritis and stated that in 75 per cent of the cases the carcinoma was secondary to some sort of gastritis. He considered polyposis, if it preceded cancer, secondary also to gastritis. Hurst[13] thought that the remainder of carcinomatous lesions of the stomach follow in 20 per cent gastric ulcer and in 4½ per cent a simple adenoma.

In 1932, Davis[5] wrote that one of the causes of primary carcinoma of the stomach had probably been discovered in chronic gastritis. He also stated that chronic gastric ulcer offered a basis for an indeterminate proportion of gastric cancers. In 1936, Eusterman[6] warned that carcinoma need not necessarily follow chronic gastritis in every instance. C. P. Baker[2] demonstrated two types of stomach mucosa and showed that there was an increase in stomach cellularity and lymphocytic infiltration together with an increase in globulin and mucous cells in the antral portion of the stomach. He has judged 3,200 cases of carcinoma of the stomach and found that in a large proportion there was a generalized hyperplasia of the mucous cells, indicating that the exciting factor must produce changes in the entire mucosa. He concluded that these changes were probably precancerous. Baker[2] has also stated that hyperplasia of the mucous cells is observed in pernicious anemia, and that this probably represented an early manifestation of the process which leads to the development of polyps and eventually carcinoma.

In 1943, W. C. Alvarez[1] reported a case in which it seemed likely that cancer was the end result of chronic gastritis. In the same year, W. C. McCarty[19] stated that carcinoma, following chronic gastritis, was a definite possibility pathologically, but that he had never seen a single carcinoma which he believed had its origin in a gastritis, that was not associated with chronic ulcer or polyp. He could see no etiologic or histologic relationship between the two conditions when a chronic ulcer was not present.

G. M. Smith[26] in 1944 reviewed the status of chronic gastritis, having stated that cancer of the stomach starts in the mucous producing cells of the stomach and goes from this focal point in all directions. He stated that Warren[30] and Collins[4] were able to trace the change in gastric epithelium from minor erosions to what was considered precancerous change present in chronic gastritis. In 1943, Guiss and Stewart[9] stated that gastric atrophy appeared to be the result of an aging organ and is not necessarily related to cancer.

In June of 1946, Maimon and Palmer[18] thought that superficial and hypertrophic gastritis might be variations of the same process rather than a distinct entity. They found atrophic changes were frequently in the upper third of the stomach and superficial and hypertrophic changes more often in the middle. Their conclusion was that regardless of the attractive theoretical considerations, the clinical significance of chronic gastritis remains unproved. In 1944, S. Warren and W. A. Meissner[30] quoted Konjetzny, Schindler, Hurst, and Judd, stating that chronic gastritis may be a precancerous lesion; however, they divided

Dr. Shellito is from the Department of Surgery, Wichita Clinic, and Dr. Stofer from the Department of Pathology, Wesley Hospital, Wichita, Kansas.

chronic gastritis into two types, the exudative type as a non-precancerous lesion, and felt that the epithelial type was a premalignant lesion. They stated that not all types of chronic gastritis need be considered precancerous lesions.

Harris[10] stated that hypertrophic gastritis may occur in a form indistinguishable from carcinoma (1945). In June of 1945, H. M. Pollard and R. R. Cooper[22] stated that hemorrhage can occur from hypertrophic gastritis and that the symptoms may be indistinguishable from those of gastric carcinoma or peptic ulcer.

The most familiar forms of gastritis could well be divided into two portions: (1) hypertrophic gastritis associated with lesions of the stomach and duodenum, and (2) atrophic gastritis associated with pernicious anemia and chronic alcoholism. There is much evidence that these two forms of gastritis may be part and parcel of the same process. It is thought by the authors that hypertrophic gastritis may well be the preliminary stage of atrophic gastritis. An obstruction of the stomach due to mechanical factors will favor the development of gastritis. The presence of gastritis does not necessarily mean that the patient will have symptoms.

Hirsh[11] was unable to find any evidence of acute or chronic inflammation in a series of stomachs taken from drunkards who died of delirium tremens.

Church, Walters, and Wellbrock,[3] in a study of stomachs resected for carcinoma, revealed that pyloric obstruction is undoubtedly a factor. In sections taken proximal to the lesion they found chronic gastritis in 92 per cent of the cases.

Fitzgerald[8] stated that it has long been the custom to associate chronic ulcer of the stomach and duodenum with persisting indigestion accompanied by periodic pain, hemorrhage, retention, etc. It is now definitely established that these signs and symptoms may occur in the absence of ulcer. Fitzgerald[8] felt that chronic gastritis was the responsible disease. He presented nine cases of "chronic follicular gastritis," which could well represent the intermediate stage between hypertrophic and atrophic gastritis, and mentioned that in one specimen it was possible to find atrophy, excessive regeneration, superficial ulceration of the mucosa, polypoid overgrowth, and hemorrhagic erosion. All of these pathologic pictures are part of one continuous degenerative and reparative process in the stomach wall. Lymphoid infiltration was a common occurrence, and, because of this finding, gastritis described by Fitzgerald was called chronic follicular gastritis. The case presented here falls into this stage of the pathologic process almost exactly (See Figures 1 and 2).

Fitzgerald[8] suggested that until the etiology of gastritis is completely known, a partial gastrectomy either of the Billroth I or modified Billroth II type should be the treatment of choice.

CASE REPORT

This 26-year-old white male presented himself complaining of stomach difficulties of one year's duration. He described this difficulty as cramps in the upper abdomen, nausea without vomiting, and a salty taste in the mouth. Indigestion, which occurred with meals or between meals, had no specific response to soda. The patient had been placed on anti-acid therapy elsewhere to no effect. The man did not drink but was a heavy smoker. The remainder of his history was essentially negative.

Gastric analysis revealed a free hydrochloric acid of 45 units with a total acidity of 52 units. There was no anemia.

The radiologist reported an indefinite lesion in the distal third of the stomach which was thought to be a neoplasm. Gastroscopy was done, and hypertrophic gastritis was seen.

Exploration was advised and carried out. The pyloric portion of the stomach was found to be indurated and almost nodular. Mesenteric nodes did not seem to be involved by tumor metastases. Upon close inspection, the distal one-third of the stomach mucosa seemed to be hyperemic, thick, and nodular. It was difficult to determine whether or not one was

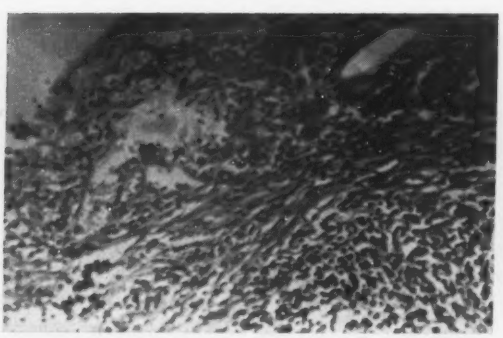

Figure 1. Chronic follicular gastritis—lymphoid infiltration.

Figure 2. Chronic follicular gastritis—polypoid growth and erosion.

dealing with a carcinoma. Frozen section examinations of the nodes and mucosa were reported as negative. A Schoemaker modification of a Billroth I procedure was done, resecting the distal one-third of the stomach. The patient made an uneventful recovery and is well one year later.

CONCLUSION

From our own experience it would seem that chronic hypertrophic gastritis, when seen through the gastroscope, is a lesion to be watched closely and to be considered as a clinical precancerous lesion until proved otherwise.

It is felt that chronic hypertrophic gastritis is one stage of a continuous cytologic process. This process might well be considered as progressive from a simple inflammatory or exudative gastritis through hypertrophic gastritis or hyperplasia, followed by atrophic gastritis and finally malignancy.

If an ulcer should appear, or a space-filling defect, either by x-ray or gastroscopic means, the patient should be explored as the only sure way to determine the malignancy or non-malignancy of a lesion in the stomach.

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Heat Stroke: Experience at the Winfield State Training School during a Record Heat Wave

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The Midwest will long remember July, 1954, because in that month it suffered one of the most disastrous heat waves in its history. For 14 consecutive days, the mercury rose to 106 degrees or higher. Uncomfortable though they were, most people survived this period with no serious effects.

Not all Kansans were so fortunate. In this state alone, Associated Press reports attributed 41 deaths to the heat. Hardest hit were the state mental institutions, where 18 patients died. At the Winfield State Training School, Winfield, Kansas, a high incidence of heat stroke created an emergency that for two days threatened the lives of more than 1,400 mental deficients. Although there were only three deaths, strenuous measures were required to prevent a major catastrophe.

At that time, the Department of Medicine of the University of Kansas School of Medicine was offering a four-months' tour of duty at Winfield as part of its residency training program. In that capacity, I suddenly had to direct on a mass scale the medical management of a disease with which I had had little previous contact. It is quite likely that other physicians, particularly those working in mental and geriatric institutions, may at some time find themselves in a similar situation. For this reason the following account of a rather unique experience with heat stroke is submitted.

PHYSIOLOGY OF HEAT REGULATION

By way of introduction, perhaps a brief review of the physiology of heat regulation would be in order. It is obvious that if body temperature is to remain constant, a balance must exist between the rate of heat production and the rate of heat dissipation. The chief avenues of heat loss are radiation, convection, conduction, and evaporation. Under ordinary conditions, the first three account for about 70 per cent, while 27 per cent is lost by evaporation of moisture from the skin and lungs. The remaining three per cent is lost in warming the inspired air and in the urine and feces.[1] However, when the external temperature reaches 95 degrees or more, evaporation becomes practically the only means of heat dissipation.[2] The efficiency of evaporative cooling depends to a large extent on the ability of the individual to secrete sweat normally.

This problem was carefully studied by Dill[3] on workers during the construction of Boulder Dam. He found that adaptation to hot climates involves not only an increase in the capacity to sweat and production of sweat of lower salt content, but also greater sensitivity in the control of perspiration.

Dill also found that it is not enough to have adequate water and salt supplied in the diet and a copious secretion of sweat. Of equal importance are the circulatory adjustments necessary for transfer of heat from areas where it is produced to the skin, where it can be dissipated. These adjustments are mediated by a temperature regulating center in the hypothalamus and consist chiefly of reflex constriction of vessels in the splanchnic bed and in other areas coincident with opening of arterio-capillary shunts in the skin. The result is a marked increase in both the quantity and rate of blood flow through the skin. At high temperatures, this may amount to a significant portion of the cardiac output.[4]

Bazett[5] has shown that during the acclimatization process there is an increase in blood volume. Hypervolemia appears to be necessary in order to compensate for the decrease in extracellular fluid caused by profuse sweating, and in order to maintain adequate

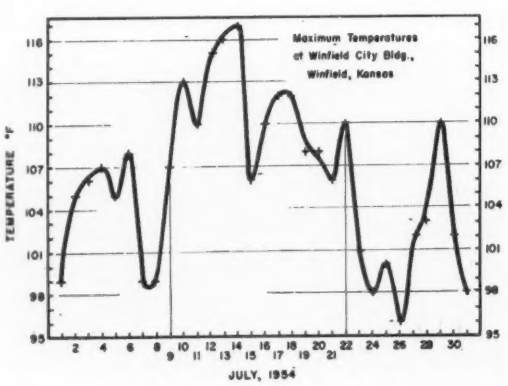

Figure 1.

Presented at the regional meeting of the American College of Physicians, Wichita, Kansas, March 18, 1955. Dr. Steinzeig is a resident in internal medicine, University of Kansas Medical Center.

circulation to the other organs when large amounts of blood are shunted to the skin.

It is not surprising that individuals who are elderly, debilitated, or dehydrated are as susceptible to hot weather as are individuals with cardiac disease or intrinsic defects of heat regulation (e.g., infants and Mongoloids).

THE EMERGENCY

In Figure 1, one can see that the month began abnormally hot. Though these readings portray conditions in a general way, the thick stone walls of the dormitory buildings made excellent insulators so that inside temperatures were actually much higher and remained high for several hours after the outside air began to cool.

By July 4, the weather began to be a subject of great concern. The Winfield State Training School was caring for more than 1,400 patients who varied in age from infancy to senescence. Most were admitted because of mental deficiency per se, although practically every type of organic brain disease and congenital malformation was represented. The population included about 200 Mongoloids in addition to a large group of elderly, debilitated patients and infants, all of whom could be expected to withstand heat poorly.

The usual prophylactic measures were strongly reinforced. Physical activity was restricted, and additional baths were given. The salt content of the diet was increased, and water carts were circulated several times a day. *Food and salt intake was excellent, but a major problem was getting sufficient fluids into the more retarded patients who simply did not drink unless the cup was held to their lips.* In the hospital building, regular dehydration rounds were made for the purpose of giving parenteral fluids to those who were not drinking enough.

No serious effects were observed until July 13, when the temperature reached 116 degrees. Late that afternoon, the first patient with heat stroke was admitted to the hospital. Before the night was over, seven more were admitted. Darkness brought little relief from the heat, and at 7:00 the following morning, the thermometer already measured 106 degrees. By mid-morning, new patients began coming in and it was clear that a full scale emergency was in progress.

A treatment center was quickly organized in the hospital building. Activities not absolutely vital to the operation of the institution were suspended so that all efforts could be directed toward meeting the emergency. Office and maintenance personnel as well as volunteer workers manned shifts around the clock to provide help where needed. The acute wards on the first floor were emptied of all patients who could safely be moved elsewhere. Long metal-topped dining room tables were set up in the main corridor. Attendants in the buildings were instructed to keep their patients as cool as possible by means of frequent showers and ice packs, and to take rectal temperatures at two-hour intervals. Patients with temperatures above 102 degrees were hospitalized immediately.

That afternoon the outside temperature reached a peak of 117 degrees, and new patients were being admitted at the rate of 10 to 15 an hour. Upon arrival, they were placed on the tables in the corridor where a rapid evaluation was made. In many cases, a good look combined with a hand on the forehead was enough to make the diagnosis. Mild cases were treated on the spot and usually responded within two or three hours. By necessity, these had to be returned to their buildings in order to make room for the new patients who were pouring in continuously. Sicker patients were taken to the acute wards where they could be given better observation and care.

The 250 non-ambulatory chronic patients housed in the hospital required constant attention because they were unable to care for themselves. Fortunately, the three nurseries had recently been air-conditioned. Otherwise these children would probably not have survived.

July 15 was slightly cooler, and with the arrival of additional help it was possible to shift the emphasis in treatment from the hospital to the dormitory buildings. By making rounds several times a day, we were able to discover and treat patients earlier, thus reducing the load on the hospital personnel who could then give closer attention to the acute and chronic wards. In this way, hospital admissions were held to fewer than five per day until July 23, when the weather finally moderated.

PATHO-PHYSIOLOGY OF HEAT STROKE

In a 14-day period, approximately 275 persons received hospital treatment in addition to an unknown number who were treated in the dormitories. Because their illness was characterized by rapidly progressive hyperpyrexia and prostration, the term heat stroke has been used. For the purpose of classification, patients with rectal temperature below 105 degrees were called mild heat stroke. Of the hospital cases, about 250 fell into this category. In addition to hyperpyrexia and prostration, examination revealed dry, flushed skin of poor turgor, parched tongue, moderate tachycardia, and moderate tachypnea.

About 25 patients had rectal temperatures between 105 and 108 degrees and were classified as having severe heat stroke. The clinical findings in one of these cases is shown in Figure 2. The three fatalities were, of course, listed in this group. One such case is

SEVERE HEAT STROKE

75 year old white male
Rectal temperature 108°
BP 160/70; Pulse 140; Resp. 40
Coma
Skin hot, dry and flushed
Dehydration
Oliguria

Figure 2.

summarized in Figure 3. It is interesting to note that all three terminated in profound peripheral vascular collapse. Our patients were not examined post mortem. However, the autopsy findings in 125 fatal cases have been described in detail by Malamud and coworkers.[6]

It would be improper to draw broad conclusions from our rather gross observations. However, some features were so constant that they deserve comment. All of our patients (except when in shock) exhibited cessation of sweating during or prior to loss of temperature control. Furthermore, as treatment was given, resumption of perspiration coincided with clinical recovery. Moderate to marked dehydration was almost always present.

Since the average healthy adult requires two to three liters of water per day to maintain normal fluid balance,[7] the amount needed to replace what is lost in sweat in high temperatures may be several times that quantity. If this loss is only partially replaced, the kidneys can continue to excrete salt in proportion to water for a time. Some depletion of both body water and electrolytes occurs, but osmotic pressure relationships are preserved. With further dehydration, the urinary output drops and the kidneys are unable to excrete enough salt with the water in the urine to prevent the extracellular fluid from becoming hypertonic. The fact that hypertonicity and hemoconcentration do occur with water depletion is demonstrated in Figure 4 by the laboratory data taken from the case used as the example of severe heat stroke (Figure 2).

FATAL HEAT STROKE

50 year old white male
Rectal temperature 108°
BP unobtainable; Pulse 140; Resp. 40
Coma, Cyanosis, Shock
Skin ashen, hot and moist
Dehydration

Figure 3.

Care must be taken to differentiate heat stroke from heat exhaustion, which differs considerably in its physiology and management. The latter occurs in individuals who replace water adequately during prolonged sweating, but who do not replace salt. These patients usually have no hyperpyrexia, but have a moist clammy skin, hypotension, and may complain of muscle or abdominal cramps.

The main features of these diseases are contrasted in Figure 5. *Our patients exhibited almost a pure form of the water depletion syndrome.* It should be emphasized that this can develop quite rapidly during a heat wave. Of course, salt depletion can also occur, but it takes longer to develop. If both are present, then treatment must be individualized with replacement of both water and electrolytes as the situation demands.

SEVERE HEAT STROKE

Serum Na	156 mEq./L.
Serum K	2.92 mEq./L.
Serum Cl	115 mEq./L.
CO_2	26.2 mEq./L.
NPN	63 mgm.
Hematocrit	58%

Figure 4.

TREATMENT

There are two primary objectives in treatment. First, the hyperpyrexia must be reduced. Since damage to tissues depends on duration as well as degree of hyperpyrexia, the temperature must be lowered as quickly as possible. There is disagreement as to how this should be done.[8] We found a safe and efficient method to consist of wrapping the patient in a wet sheet covered with a thin layer of crushed ice. This provides cooling effect both by conduction and by evaporation. In our experience, this was considerably faster than sponging or cold baths. The method was applied more than 1,000 times without apparent ill effect.

Secondly, dehydration must be corrected. If used cautiously, the intravenous route is preferable. However, one must be careful not to overload the already straining heart. We used hypodermoclysis almost exclusively because it is so much easier to administer and requires practically no trained supervision. The preferable repair solution in water depletion is 5 per cent glucose in distilled water. Saline is indicated only

HEAT STROKE	HEAT EXHAUSTION
Water depletion syndrome	Salt depletion syndrome
Hypertonic extracellular fluid	Hypotonic extracellular fluid
Intracellular dehydration	Intracellular edema
Terminal circ. collapse	Early circulatory collapse
Treatment: Water, 5% Glucose	Treatment: Saline, Potassium

Figure 5.

if there is also salt depletion. As soon as the patient is able to drink, he can then be given additional fluids by mouth.

Aspirin was given emperically and appeared to be beneficial. The drug is thought to have an effect on the central temperature regulating center in addition to its properties as a peripheral vasodilator. We gave adults a single dose of 15 grains orally. Children were given one grain per kilogram of weight.

The basal metabolic rate rises about 7 per cent for each degree Fahrenheit of body temperature elevation. Since this results in a marked increase in oxygen consumption, we administered oxygen by nasal catheter to the limit of our equipment.

SUMMARY AND CONCLUSIONS

1. Experience with a form of heat stroke under unusual circumstances is described.

2. Dehydration with extracellular hypertonicity and early cessation of sweating would appear to be extremely important factors in the pathogenesis of heat stroke. These events occurring in a susceptible population very likely explain the high incidence of the disease at Winfield.

3. Three deaths were due to acute water depletion rather than to salt depletion such as would be found in chronic heat exhaustion.

4. Fluid replacement should be accomplished parenterally and with 5 per cent glucose in water, unless an electrolyte deficiency also exists.

5. Body temperature can be quickly and safely reduced by wrapping patients in wet sheets covered by a thin layer of crushed ice.

ACKNOWLEDGEMENT

The author would like to express his deep admiration for the staff and personnel of the Winfield State Training School as well as for the many volunteer workers whose collective efforts were responsible for the successful termination of the emergency. Thanks should also go to Miss Leitha Bunch of the Snyder Foundation, Winfield, for the laboratory data, and to Mr. Charles O. Booth of the *Winfield Daily Courier* for supplying weather information.

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One of the weaknesses of this generation is fear. Fear of atomic destruction, fear of cancer, fear of heart attacks, and other fears daily plague the minds of our people. Unless checked, this fear-complex may cause untold harm. As counselors, physicians have an excellent opportunity to combat this epidemic of fear. In the office, at the bedside, through health forums, radio programs, TV shows, and emergency services doctors can alleviate to a great extent fear of dreaded diseases and sudden sickness.

Dan Mellen, M.D., President
Medical Society of the State of New York

Symptoms from Thymic Enlargement in Young Infants

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The enlarged thymus gland as a cause of respiratory disease has been a topic of controversy since early times. In 1830 Kopp presented the theory that there was an etiologic relationship between the hyperplastic thymus and laryngospasm.[1] He introduced the term "thymic asthma" which was widely accepted until Friedleben, in 1858, completely refuted this concept with his anatomic studies.[2]

In 1889 Paltauf described a condition that he believed predisposed to stridor and even to sudden death in children.[3] This he named the "lymphatic constitution." He believed stridor and sudden death were caused by pressure of an enlarged thymus on the large mediastinal vessels and nerves and on the trachea.

As time passed, the role of the enlarged thymus in sudden death received more emphasis, and gradually the term "status thymicolymphaticus" came into being. This was an indefinite syndrome, but it became quite popular and was commonly labeled the cause of death in many infants who died under questionable circumstances.

The studies of Hammar[4] and Boyd[5] and others have shown, however, that the normal thymus varies greatly in weight at birth and that the thymus persists throughout life with a wide range of weights at all ages. It has been proved beyond question that thymus glands that are heavier than 20 grams need not be pathologic but rather represent normal structures which have not yet undergone atrophy.[5] The belief that hyperplasia of the thymus gland is the cause of sudden, unexplained death has diminished markedly since demonstration of the wide variation of normal thymic weights. The studies of Hammar and Boyd have made invalid the anatomic diagnosis of "status thymicolymphaticus."

Occasionally infants are seen who have a variety of symptoms that might be explained on the basis of pressure of an enlarged thymus on surrounding structures. Symptoms thought to be due to some form of respiratory obstruction are many and varied and in the past often were grouped under the term "thymic stridor." Some of these symptoms include crowing, coughing, choking, dysphagia, pallor, asthmatic wheezing, and even shortness of breath and cyanosis. If any of these symptoms were accompanied by roentgen findings of a widened mediastinal shadow, this was taken as evidence that thymic enlargement was the cause of the patient's symptoms.

However, with more modern diagnostic techniques, these symptoms can usually be demonstrated to be due to other causes, such as congenital heart disease, anomalies of the aortic arch and its branches, anomalies of the larynx and epiglottis, foreign bodies, atelectasis, and infectious or neoplastic lesions of the respiratory tract. These non-thymic factors usually are given prior consideration by most physicians before seriously considering the thymus as the cause.

Occasionally, however, infants are seen who have symptoms which cannot be accounted for by non-thymic factors and who have an enlarged thymic shadow on roentgenograms. Some of these infants have been found to have a rapid and dramatic disappearance of symptoms and shrinking of thymic shadows following irradiation over the thymic area. In the past five years, four such infants have been observed on the Pediatric Service at the University of Kansas Medical Center. The clinical courses of these four infants are presented in this report.

CASE 1

Case 1. K. G., a three and one-half months old colored male, was admitted to the University of Kansas Medical Center with the chief complaint of a harsh cough that had been associated with crying and feeding since the age of four weeks. The infant was said to have coughed daily since coming home from the hospital at one month of age. Four days prior to admission the baby had a severe crying spell, the coughing became worse, and the baby had difficulty breathing. Because the cough continued, the baby was brought to KUMC and was admitted for study.

The mother stated that the extremities of the patient became darker from time to time without apparent cause, even when the baby was not crying. No fever or vomiting had been present and the weight gain had been adequate.

The infant was the product of a full term, normal delivery with a birth weight of 10 pounds, 14 ounces. An omphalocele was repaired three hours after birth, and the patient remained in the hospital for the first month of his life.

From the Department of Pediatrics, University of Kansas School of Medicine, Kansas City, Kansas.

Physical examination revealed a well developed (25 pounds), obese, three and a half-months old male who was alert and in no obvious distress. Examination of the head was negative, and the anterior fontanel was normal in size and tension. The eyes, ears, nose, and throat were normal in appearance. Respirations were noted to be 48 per minute and exaggerated, but no wheezing, crowing, or retraction was observed. The baby would cough occasionally, and when he cried this cough became almost continuous. Both sides of the chest expanded equally, but the breath sounds were suppressed over the right upper anterior chest compared to the left side. Dullness to percussion could not be made out. The remainder of the lung fields seemed to be normal, and the breath sounds were vesicular. The heart sounds were of good quality, and normal sinus rhythm was present. No cardiac enlargement or murmurs were found. Examination of the abdomen was not remarkable except for a transverse scar, the result of umbilical surgery at birth.

Laboratory tests showed the urine to be normal and the red blood count to be 4.62 million cells per cu. mm. with a hemoglobin of 10.8 grams (70 per cent). The white cell count was 10,750 per cu. mm. with 24 per cent polys, 72 per cent lymphocytes, 1 per cent eosinophils, and 3 per cent monocytes. Nose and throat cultures showed the usual flora. An electrocardiogram was normal. A chest x-ray, including postero-anterior, lateral, and fluoroscopic examination showed that the mediastinum was unusually wide. It was felt that this mass represented an enlarged thymus. The remainder of the x-ray was entirely normal.

The patient received two irradiations, a day apart with a total of 296 r. It was noted that symptoms began to disappear the day after the second treatment and the coughing became minimal. The diminution of breath sounds in the anterior right chest that had previously been present was gone the following day, and auscultation revealed the breath sounds equal bilaterally. X-rays taken four days after the last treatment showed a marked decrease in the size of the mediastinal mass. The baby was dismissed from the hospital on the ninth hospital day.

He was returned to the Out-patient Clinic for follow-up in one month. The parents reported that the baby had had no more of his previous symptoms; the cough did not reappear, even with hard crying. Physical examination was entirely negative, and chest x-rays taken on this date also showed that the mediastinal shadow had not returned.

CASE 2

Case 2. F. W., a seven months old white male, was admitted to KUMC with the chief complaint of respiratory difficulty since the age of one month. The patient was the product of a full term, spontaneous delivery with a birth weight of 7 pounds. At birth the cry was immediate, and there was no respiratory distress or cyanosis. At one month of age the child began having noisy respirations that were first thought to be an upper respiratory infection. However, this symptom persisted and breathing was at times described as "bubbling" in type. He had never been cyanotic or actually short of breath, but when he cried, he frequently made a crowing sound.

During the following months the baby had several upper respiratory infections, and the above symptoms increased and were accompanied by a severe cough. The baby was started on strained foods at three months of age. However, during the month before admission to the hospital he refused all solids, and the mother stated he seemed to have difficulty swallowing them. He was referred to KUMC because of the persistence of noisy respirations and his refusal to eat solid foods.

On admission the patient was a well developed, well nourished, seven months old boy who did not appear acutely ill, but his loud breathing could be heard several feet away. There was a stridor when the baby cried. The neck was short in appearance, and there seemed to be an increase in the amount of soft tissue. The lungs were negative to percussion, and no increase in sternal dullness was noted. No rales were present, but rhonchi were heard throughout the upper lung fields and were thought to be transmitted from the upper respiratory passages. The heart was normal with no enlargement or murmurs. The remainder of the physical examination was entirely normal for a boy of this age.

The laboratory reported the urinalysis as normal; red blood cell count was 3.87 million per cu. mm. with hemoglobin 9.8 grams (64 per cent); white blood cell count was 16,200 per cu. mm. with 21 per cent polys, 70 per cent lymphocytes, and 9 per cent monocytes. Nose and throat cultures showed the usual flora.

X-rays of the chest revealed a shadow in the superior, anterior mediastinal area which was diagnosed as enlarged thymus. Direct laryngoscopy showed no abnormalities, and the nasopharynx was described as clean on palpation except for the presence of a small adenoid pad. The thymic area was irradiated twice with a total of 214 r. It was noted that respirations became much quieter by the day following the second irradiation, and no more wheezing or stridor was noted. Repeated chest films and fluoroscopy in five days showed that the upper anterior mediastinal shadow had decreased markedly in size. The baby was dismissed eight days after admission to be followed by his local physician.

The patient returned to KUMC for follow-up in

six months, and he had had no recurrence of his respiratory symptoms and was eating all solids without difficulty. X-ray examination at that time showed no enlargement of the mediastinum, as was described earlier.

CASE 3

Case 3 L. G., a three and one-half months old white female, was well until one and one-half months of age when she developed a "cold" with nasal discharge and difficult breathing. Respirations were labored with retraction of the chest accompanied by inspiratory wheezing. No fever was present. The nasal discharge cleared up in two days, but stridor continued and the mother noticed that it seemed worse when the baby was lying down. Subsequently the position made little difference and the stridor was present most of the time. The child was treated with penicillin and steam inhalations, but without relief of symptoms, and was admitted to KUMC because the stridor seemed to be increasing. No cyanosis was noted and the cough was minimal. The baby had a good appetite and was gaining weight well.

The birth and developmental history were normal and the family history was non-contributory.

The patient was a well developed, well nourished, white female, three and one-half months of age, who did not appear chronically or acutely ill. The chest had a normal contour, but there was marked inspiratory sternal retraction. Breathing was stridorous with a raspy, coarse, inspiratory sound. Breath sounds were not unusual and no rales were heard. There was no dullness to percussion. Examination of the heart revealed normal sinus rhythm, heart sounds were strong, and no murmurs or thrills were present. Examination of the abdomen, genitalia, head, eyes, ears, nose, and throat was not remarkable.

Laboratory results were as follows: urine was normal except for 2-3 pus cells per low power field; red blood cell count was 4.22 million per cu. mm.; hemoglobin was 10 grams; white blood cell count was 9,600 with 17 per cent polys, 73 per cent lymphocytes, 4 per cent eosinophiles, and 5 per cent monocytes. Tuberculin and histoplasmin skin tests were negative. Direct laryngoscopic examination revealed no abnormal findings. Fluoroscopy and x-rays of the chest showed a soft tissue structure that filled the anterior mediastinum and was separate and distinct from the heart shadow. This mass was interpreted as an enlarged thymus.

The superior mediastinum was irradiated on two occasions with a total dose of 206 r. Repeat x-rays of the chest five days later failed to reveal the soft tissue structure that had been visualized previously. The nursing and house staff noted a marked improvement in the baby's respirations, and the stridor that had been present for one and a half months was now absent. The baby was dismissed, and communication with the family physician in six months revealed that the girl had done well with no return of stridor or respiratory wheezing.

CASE 4

Case 4. D. D., a white male, four and one-half months of age, was admitted to KUMC with the chief complaint of crowing and respiratory wheezing. This stridor had been noted first at approximately three months of age and seemed to be worse at night or when the baby was on his back. The wheezing was stated to be present almost constantly, occasionally accompanied by a cough. He also frequently "gagged and coughed while eating" but he had gained weight well. Three weeks before admission to the hospital the patient had an episode during which he was reported to have become cyanotic following a severe coughing spell.

The baby was the product of a full term, normal delivery with a birth weight of 7½ pounds. At three weeks of age the baby started vomiting, which shortly became projectile in type. An operation for pyloric stenosis was performed when he was five weeks of age.

This baby, 4½ months old, was well developed and well nourished and weighed 15 pounds, 2 ounces on admission. His chest appeared normal in contour, and expansion was equal bilaterally. The breathing was noisy and wheezing in type, and when the baby was upset there was definite inspiratory substernal retraction. Breath sounds were heard throughout with scattered rhonchi over the entire lung fields. There was no dullness to percussion. No murmurs were heard. The remainder of the physical examination was not remarkable except for a midline epigastric scar.

The laboratory reported the following results: urine was normal; red blood cell count was 3.92 million per cu. mm.; hemoglobin was 9.7 grams; white blood cell count was 13,500 per cu. mm. with 41 per cent polys, 56 per cent lymphocytes, and 3 per cent monocytes. Nose and throat cultures showed the usual flora. Direct laryngoscopic examination revealed no abnormal findings.

Fluoroscopy and x-ray examination of the chest showed a shadow in the mediastinum that was located anteriorly. It was the impression of the roentgenologist that the mass was thymus. A series of three x-ray treatments was given over the thymic area with a total dose of 270 r. Following this moderate irradiation, re-examination of the chest by x-ray six days later revealed a marked decrease in the size of the thymus shadow. It was noted clinically at this time that the patient was breathing much easier, and he was sent home. A follow-up examination was not obtained.

COMMENT

The production of symptoms by an enlarged thymus is probably a rare event. More than 60,000 admissions to the Pediatric Out-patient Clinic and Children's Pavilion occurred in the five years from 1949 to 1954, and the four cases presented here were the only patients suspected of having symptoms related to an enlarged thymus. The four patients form only a small part of the large number of children admitted with similar complaints that were thought to be caused by other factors, and they also make up only a small fraction of the infants who had an enlarged thymus by x-ray examination but were without symptoms.

Roentgen diagnosis of thymic enlargement is still unsatisfactory since standards for normal limits at various ages have never been definitely established. Furthermore, the width of the supracardiac shadow varies in the same person under different conditions such as respiratory phase, cardiac diastole, position of the patient, and increased intra-abdominal pressure. Pancoast's technic for showing encroachment of the thymus on the trachea added little to the exact determination of the size of the thymus because of lack of satisfactory standards for the size and variation of the normal tracheal lumen.[8] Tracheal encroachment has been reported in more than 50 per cent of young children who were asymptomatic.[19]

Small amounts of roentgen therapy were administered to each of the four infants in this report as a therapeutic trial after the other likely causes had been considered and dismissed. The diagnosis of thymic enlargement as a cause of the symptoms was not entertained until it had been demonstrated that symptoms cleared in two or three days after irradiation. Had the symptoms not cleared, further diagnostic tests of a more difficult nature would have been undertaken, including bronchoscopy and perhaps bronchography.

It was felt that a small amount of irradiation was justified as a therapeutic trial before attempting more risky endoscopic procedures. It should be noted that all of these infants had direct laryngoscopy prior to irradiation. This procedure is comparatively easy and safe in infants and can be of considerable diagnostic help when performed by a skilled operator.

The rapidity with which symptoms have cleared following irradiation of the thymus has led to the obvious suggestion that mechanical factors causing pressure on mediastinal structures were responsible for the symptoms. This apparently reasonable assumption probably should not be accepted without some reservations in view of the limited knowledge of the mechanical factors and in view of alternative theories.

Two decades ago Aldrich suggested that such respiratory symptoms might be explained by increased vagal activity causing spasm of smooth muscle of the respiratory tract.[12] More recently Allen and his group have made a similar proposal.[21] While neither of these reports produced any conclusive evidence in support of their hypothesis, it is important to realize that alternative proposals have been made and their validity is no less acceptable than the long held view concerning mechanical obstruction by an enlarged thymus.

Sudden deaths do occur in young infants. Postmortem studies carefully done on large series of such infants have revealed a surprisingly high incidence of infection as a cause of death and have failed to substantiate "status thymicolymphaticus" or enlarged thymus as an acceptable cause of death.[15, 16, 17, 18, 20]

SUMMARY

Case histories of four infants with respiratory symptoms who received roentgen therapy to the thymic area have been reported. The relationship of thymic enlargement to production of symptoms has been discussed. Proof that thymic enlargement causes symptoms is circumstantial.

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PRESIDENT'S PAGE

DEAR DOCTOR:

There is only one reason for the existence of a medical journal and that is the publication of worth-while scientific articles. The JOURNAL OF THE KANSAS MEDICAL SOCIETY is our official publication for the minutes of our meetings, it carries notice of deaths of our members, stories on county societies and individual activities, and it even has a President's Page read by some 10 to 20 faithful readers. All of these are ancillary to the prime purpose which is the dissemination of medical knowledge.

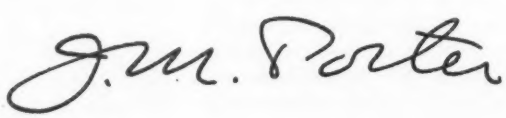
I like to think of our editorial board as men with heavy glasses and worried looks surrounded by a mass of manuscripts, rejecting nine out of ten papers submitted to them. Unfortunately, this is not the case. The JOURNAL OF THE KANSAS MEDICAL SOCIETY desperately needs good material. The backlog of articles which permits variety, editorial discrimination, the publication of timely or seasonal papers, and the like, simply is not there any more.

Why this is true—the multiplicity of journals, the lost itch of the Kansan to burst into print, the failure of speakers to make their contributions exclusive—we will leave to Orville Clark and his efficient staff. The correction of this situation, however, is an urgent problem for every one of us who reads the JOURNAL. You and I must either write articles or we must see that others write them.

First, we must see that interesting material is collected and written up. This can consist of formal papers on general or specialized subjects, simple case reports, statistical analyses, or even discussions of the social, economic, or political aspects of the practice of medicine. Members of the Kansas Medical Society, guest speakers at our larger meetings, youngsters doing research, and old-timers viewing with alarm are all eligible. Once these are written in reasonably good English in a form that a printer can read, they must be submitted for publication. You and I must see that these steps are taken.

To bring this point home I am asking Pauline Farrell to send proofs of this page to all officers and councilors of the Kansas Medical Society and to secretaries of the larger county and district organizations. Thus, even though they regularly skip the President's Page, the matter will be brought to their attention. Later, we may ask for a report as to what has been accomplished.

The burden still falls on the individual member of the Kansas Medical Society. What can you do to help your JOURNAL?



EDITORIAL COMMENT

A Universal Fable

There is an ancient legend which tells us that when a man first achieved a most notable deed he wished to explain to his tribe what he had done. As soon as he began to speak, however, he was smitten with dumbness, he lacked words, and sat down. Then there arose—according to the story—a masterless man, one who had taken no part in the action of his fellow, who had no special virtues, but afflicted—that is the phrase—with the magic of the necessary words. He saw, he told, he described the merits of the notable deed in such a fashion, we are assured, that the words "became alive and walked up and down in the hearts of all his hearers." Thereupon, the tribe seeing that the words were certainly alive, and fearing lest the man with the words would hand down untrue tales about them to their children, they took and killed him. But later they saw that the magic was in the words, not in the man.

This was written by Rudyard Kipling, but the fable has a sequel which is not written by anyone. What happened next does not need to be recorded. It is fresh in everybody's memory. It is a universal fable.

The tribe soon learned they could not put to death these words for they skipped endlessly about the village. Then they saw to their sorrow that the man had not been killed either because not only were his magic words alive but now the notable deed he had described was coming to be known as his accomplishment.

The man who first achieved this deed and who was smitten with dumbness pondered the lie sadly, and at last he found his tongue to tell the tribe the true story in his own crude words. When he sat down the tribe shouted that they did not believe him, and they killed him for telling untrue tales and for boasting about himself.

He was quickly forgotten because there arose in the tribe yet another man who had a strong voice. He said, "Do you remember how the story went? Listen, I will tell it to you again." And even though the whole tribe knew this story by heart they marveled as he told it over and over and over again. He did nothing else but repeat the story of this notable deed and, in truth, the tribe made him its chieftain.

From that day to the present the chieftain made the laws and caused the tribe to bow to his wishes. He, with the magic story, now controlled their lives and later, when the tribe once again discovered that the magic was in the words, not in the man, it was too late.

What's Your Pet Peeve?

With the thought in mind that "audience participation" might make the JOURNAL of more interest to its readers, the Editorial Board has considered many suggestions for columns in which members of the Kansas Medical Society might express themselves. Valid arguments against one idea after another have deterred action by the Board, and few members of the Society have written anything for the JOURNAL other than an occasional scientific paper or a rare editorial.

At a recent meeting the Board considered a suggestion that we inaugurate a "Pet Peeves" column, written by readers of the JOURNAL. From association with physicians over the course of the years, it seems obvious to this writer that most of us have material in mind that could easily be transferred to paper and mailed to the JOURNAL.

It is natural that we are irritated by the peccadillos of others. And it's just as natural that some of our habits are annoying to our associates. Or perhaps it's just a situation that gets star billing on an individual's list of pet peeves. Everyone seems to have something that acts as an abrasive on his tranquillity. We believe a column devoted to the sources of irritation would be interesting, and an exchange of ideas might even result in more harmonious relationships.

It was decided, therefore, that the JOURNAL will begin such a column and will continue it as contributions are received. The wishes of those who prefer to remain anonymous will be respected. Articles may be long or short, as the authors desire, and there are no restrictions on subject matter or style. The Editorial Board will look forward to learning the answer to the question, "What's your pet peeve?"

To start the ball rolling, we print below an example that was written in colloquial style by Dr. Peter V. Siegel for publication in *Missouri Medicine* in a column headed "Crossroads Comment":

"Suppose most folks sometime or other wish old Alex hadnt invented the telephone. Today was one of those days when I just couldnt get anything else done except answer. Always something different though, thats what makes it interesting. Case you sometimes don't answer you always end up wondering who it was and if you were missing something. I have read and heard about all manner of ways of handeling the telephone problem but none of them work out excepting answering yourself.

"It would appear that frequently we might all brush up on our telephone manners. There is such a thing and it requires a little more effort than when you are meeting somebody face to face. Bet it never

occurrs to some folks that they are very discurteous over the phone.

"A ferinstance. Today was quite an occasion in my book. I had a patient referred to me by a specialist in the city. That happens about as often as hell freezes over and they dont get many cold snaps down there. The referral came via the phone. His secretary placed the call. She acted like I didn't have anything else to do but sit by the phone and wait for it to ring so I should have been there but I wasnt. Reckon about that time I was way up on Haw Creek making a call. Anyhow by the time I got to the hospital the operator had been calling every three minutes. After we made connections the secretary said wait a minute, Doctor wants to talk to you. It could have been most any of seven hundred and eighty five so all I had to do was wait and see. Six minutes later she came back to see if I was still waiting and to reassure me I would only have to wait a minute more.

"Right then is when it happened. At any rate when I place a call I make durn sure the party Ime calling doesnt have to wait on me. I just might be asking a favor of him. I dont think its at all unreasonable to expect that little courtesy when someone calls me. Then too its mighty nice to know who you are talking to. That little problem will be solved when we have teleTV or something.

"But wouldnt it be an awful state of affairs if Alex hadent invented the telephone. Incidentally we still have some of his first models in use out here at the Crossroads."

Daydreaming

Among the more restful summer diversions is daydreaming. As a substitute for travel it has the advantage of saving expense as well as time. It spares the discomfort of driving or missing scheduled services. You escape carrying suitcases, tipping bellboys, fighting mosquitoes. You can avoid social entanglements with boring people.

Daydreaming has other advantages also. Your casting is superb, the fish strike, and you land beautiful fish one after another. The mountains are clear and cool, and at the seashore you tan a golden brown and never burn at all. The cities are fabulous and filled with endless excitement.

But you can daydream better than that. You can conjure a series of secret adventures of any kind your mood advises and live them through without moving from your chair.

You are not confined to this earth but can wander among the planets and see the universe from the vantage of far away places. Read in a book on astronomy about Betelgeuse or Antares, a couple of the giant stars that dwarf our sun. How large are they?

Put our sun at the center of Antares. Then swing the earth in its orbit at a radius of some 93 million miles from the sun and you still have several million miles to spare before you touch the circumference of this star! And this is but one of countless bodies flying through space so far away that light takes years or thousands of years to traverse the distance.

Does that give you room for your imagination? Does the magnitude of creation leave you with a feeling of awe that among the myriad masses swinging around in space and after the eons while they were exploding and cooling you stand on this particle of dust today to view the universe about you?

What now, little man, of your atom bomb and your income taxes and your right-to-work and your bikini bathing suits and your social security and your withering front lawn? Stand on this precipice of time and space and contemplate the wonder of your existence, of why you are here and what you have done of significance.

But now it ceases to be recreation for a summer day and we are breathing upon words whose meaning we do not understand. . . .

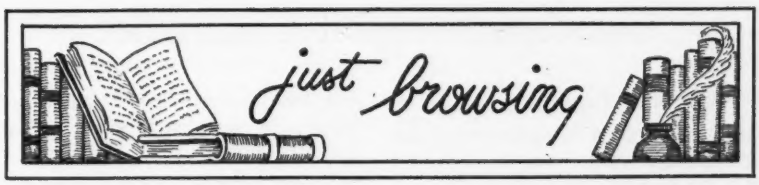
A.M.A. Publications

Dues-paying members of the American Medical Association may receive any one of nine specialty journals, in lieu of the *Journal of the American Medical Association,* should they wish to do so. Although this represents no change in A.M.A. policy, the provision for substituting one subscription for another is reviewed here because some confusion still exists.

Any physician who pays dues to the A.M.A. is entitled to receive, as part of his membership benefit, the *Journal of the American Medical Association.* Many specialists, however, find other A.M.A. publications of more value. As a courtesy to those who prefer specialty journals, the A.M.A. has listed the following publications which may be received instead:

A.M.A. Archives of Internal Medicine
A.M.A. American Journal of Diseases of Children
A.M.A. Archives of Dermatology
A.M.A. Archives of Neurology and Psychiatry
A.M.A. Archives of Pathology
A.M.A. Archives of Surgery
A.M.A. Archives of Otolaryngology
A.M.A. Archives of Ophthalmology
A.M.A. Archives of Industrial Health

A physician who wishes to make a change in the publication he receives should write the Membership-Circulation Department, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois. In addition to information about the journal he wishes to receive, he should give the name of the publication he has been receiving and the name of his state society.



"I believe it has not yet been published that certain chronic affections of the skin of the nipple and areola are very often succeeded by the formation of scirrhous cancer in the mammary gland. I have seen about fifteen cases in which this has happened, and the events were in all of them so similar that one description may suffice. . . . In all of them the disease began as an eruption on the nipple and areola. In the majority it had the appearance of a florid, intensely red, raw surface, very finely granular, as if nearly the whole thickness of the epidermis were removed; like the surface of very acute diffuse eczema, or like that of an acute balanitis. From such a surface, on the whole or greater part of the nipple and areola, there was always copious, clear, yellowish, viscid exudation. The sensations were commonly tingling, itching, and burning, but the malady was never attended by disturbance of the general health. I have not seen this form of eruption extend beyond the areola, and only once have seen it pass into a deeper ulceration of the skin after the manner of a rodent ulcer.

"In some of the cases the eruption has presented the characters of an ordinary chronic eczema, with minute vesications, succeeded by soft, moist, yellowish scabs or scales, and constant viscid exudation. In some it has been like psoriasis, dry, with a few white scales slowly desquamating; and in both these forms, especially in the psoriasis, I have seen the eruption spreading far beyond the areola in widening circles, or, with scattered blotches of redness, covering nearly the whole breast.

"I am not aware that in any of the cases which I have seen the eruption was different from what may be described as long-persistent eczema, or psoriasis, or by some other name, in treatises on diseases of the skin; and I believe that such cases sometimes occur on the breast, and after many months' duration are cured, or pass by, and are not followed by any other disease. But it has happened that in every case which I have been able to watch, cancer of the mammary gland has followed within at the most two years, and usually within one year. . . .

"The formation of cancer has not in any case taken place first in the diseased part of the skin. It has always been in the substance of the mammary gland, beneath or not far from the diseased skin, and always with a clear interval of apparently healthy tissue.

"In the cancers themselves, I have seen in these cases nothing peculiar. . . .

"The single noteworthy fact found in all these cases is that which I have stated in the first sentence, and I think it deserves careful study. For the sequence of cancer after the chronic skin-disease is so frequent that it may be suspected of being a consequence, and must be always feared, and may be sometimes almost certainly foretold. . . .

"In practice, the question must be sometimes raised whether a part through whose disease or degeneracy cancer is very likely to be induced should not be removed. In the member of a family in which cancer has frequently occurred, and who is at or beyond middle age the risk is certainly very great that such an eruption on the areola as I have described will be followed within a year or two by cancer of the breast. Should not, then, the whole diseased portion of skin be destroyed or removed as soon as it appears incurable by milder means? I have had this done in two cases, but I think, too late. . . ."

So wrote Sir James Paget in 1874 in describing the disease which still bears his name. In the ensuing 80 years little has been added to the original description except the study of microscopic slides and controversial theories of the origin and nature of the disease. This paper, only 1050 words in length (of which over half is reproduced here), has been described by Boyd as "one of those masterpieces which were commoner in the nineteenth than the twentieth century." Accurate observation and careful recording of facts can demonstrate much without the use of fancy gadgets or elaborate laboratory procedures.—*O.R.C.*

Clinicopathological Conference

CASE PRESENTATION

This was the first K.U. Medical Center admission for this 74-year-old white male, who was admitted on November 15, 1954, and died 12 days later.

The patient was deaf, senile, and uncommunicative at the time of admission, and no history was obtainable from him. A son stated that the patient was relatively well until 1951, at the time of the flood, when he had an illness diagnosed as pneumonia. He was seen by a local physician who treated him with penicillin. After a somewhat protracted convalescence of six to eight months, the patient recovered.

He remained fairly well until November, 1953, at which time he developed an illness characterized by weakness, weight loss, and a productive cough which was again diagnosed as pneumonia. The patient was treated with penicillin and initially responded to the treatment, but he did not return to the state of health that was his prior to the 1953 illness. Since the onset of the illness, the patient had remained at home, practically bedridden. He had a productive cough, bringing up an undetermined amount of foul-smelling sputum. Approximately one week prior to his admission to KUMC he was hospitalized elsewhere, where one sputum smear was reported as positive for acid-fast bacilli, and the patient was transferred to the K.U. Medical Center.

The patient's relatives denied that the patient had had any previous illnesses or surgery. He had been a farmer, later a railroad worker, and finally had retired.

The patient had one sister who died of tuberculosis when approximately 30 years old, some 30 years ago. There was no other significant family history.

System review revealed only a chronic productive cough, anorexia, and a progressive weight loss of approximately 35 pounds during the past year. He had been a smoker for many years, but relatives denied that the patient consumed alcoholic beverages excessively.

Physical examination revealed an emaciated, elderly, chronically ill white male who was alert to his surroundings but who would not talk and did not understand well. He coughed intermittently during the examination. Fundoscopic examination revealed grade I arteriosclerotic retinopathy. The patient was edentulous. There was no significant lymphadenopathy. There was an increase in the anterior-posterior diameter of the chest with dullness over the left posterior chest base, decreased breath sounds, and absent tactile fremitus in this area. Bronchial breathing was heard over the right upper lobe. Numerous inspiratory rales were heard throughout both lung fields. The point of maximum impulse was not determined. Heart tones were distant, but there was a normal sinus rhythm and no thrills or murmurs. Fingers appeared slightly clubbed. The rest of the physical examination was not contributory.

Hemoglobin on admission was 11.5 grams or 74 per cent, with a hematocrit of 42.5 cc. White count was 13,750 with 69 per cent polys, of which 66 were filamented and 3 non-filamented. White count one week later was 19,200 with 74 per cent polys. Hemoglobin and hematocrit remained unchanged during the hospitalization. VDRL (serology) was non-reactive. Fasting blood sugar was 102 milligrams per 100 cc. Chlorides were 98 mEq./liter and CO_2 was 36.6 mEq./L. Carbon dioxide four days later was 33.8. Nonprotein nitrogen was 38 and total cholesterol was 140 mgm./100 cc. Skin tests on two different occasions were negative with tuberculin, histoplasmin, blastomycin, and coccidioidin. Sputum culture revealed *Hemophilus influenzae,* highly sensitive to Terramycin and Chloromycetin. Sedimentation rate was 23 mm. in 30 minutes, and 25 mm. in one hour. Blood pH November 24 was 7.34. Three sputums were negative for acid-fast bacilli on smear.

For the first six hospital days, the patient's temperature did not rise over 99.6 degrees. On the seventh hospital day, a fever spike to 103 degrees occurred, subsiding the following day. Temperatures thereafter ranged between 100 and 101 degrees. On the fifth hospital day the patient began receiving Terramycin and postural drainage three times a day. The patient remained relatively uncommunicative during his hospitalization. He ate fairly well and was ambulatory for 20 minutes twice a day. His state of hydration did improve initially; however, on occasion supplementary parenteral fluids were needed. On November 26, 1954, a left supraclavicular node biopsy was done. The pathological report was acute lymphadenitis with slight serous atrophy of fat.

The patient was found dead in bed at 1:00 a.m. on November 27, 1954.

Question: During the time the patient had the protracted illness, did he have a productive cough and was he ever febrile?

Dr. Dowel (resident in medicine): I don't have that information.

Question: Following the lymph node biopsy, was his course the same? Was the patient given oxygen after this?

Edited by Glen R. Shepherd, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, surgery, radiology, and pathology of the University of Kansas Medical Center as well as by the third and fourth year classes of medical students.

Dr. Dowel: No, he was not. There was no essential change in his condition after the biopsy.

Question: Were any cystological studies done on the sputum?

Dr. Dowel: No.

Question: Did the patient have any pain or was he dyspneic?

Dr. Dowel: He had no pain, and while at rest he was not dyspneic at all. I must admit that we did not exert him at all. He was rather tired after going through the morning routine: eating, taking a bath, and postural drainage. He would begin to perk up around noon and feel better.

Question: Did he have any hemoptysis?

Answer: No.

Question: Was the sputum production continuous or did he have intermittent episodes of productive sputum?

Answer: He began producing foul-smelling sputum in 1953.

Question: Could you give us an idea of the amount of this sputum?

Dr. Dowel: When he coughed in the hospital, one to two ounces a day was raised. This varied from day to day. On some days he had difficulty raising sputum.

Question: How long had this patient been non-communicative?

Answer: The patient actually had a hearing aid, but we could never get it to work while he was in the hospital.

Mr. Gene Schillie (student)*: This electrocardiogram shows a sinus rhythm and rate of approximately 75, although there are some irregularities. The P-R interval and the QRS complexes are normal. The RST segment is isoelectric. Flattening of the T wave in V_1, and an inversion of the T wave slightly in V_4, V_5 and V_6 suggest that this patient had myocardial ischemia and also a low voltage. The other electrocardiogram is similar.

Dr. Delp: Dr. Lin, you saw this electrocardiogram originally. At that time you commented about the lack of an R spike in lead III. You suggested the possibility, and asked for a repeat, that this patient might have a constrictive pericarditis. Do you think that still might occur to you?

Dr. Lin: That is a possibility, and so is myocarditis.

Dr. Jackson: This tracing with its low voltage is quite possibly due to myocarditis. It is quite possible that there is something to cause a lot of potential to go out across the heart, such as pericarditis for instance, constrictive or otherwise. However, I don't think this tracing can give you that diagnosis.

Dr. Delp: You could be helpful if you said it looks like pericarditis. Does it?

Dr. Jackson: No, I wouldn't go that far, but it is a low voltage and could be compatible with pericarditis.

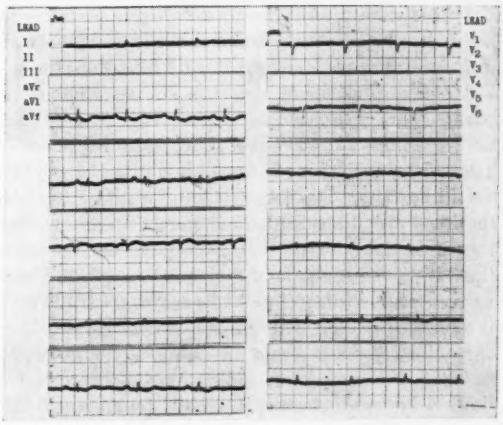


Figure 1. Electrocardiogram taken November 17 and discussed in text.

Dr. Germann (radiologist): The chest film made November 18 showed deviation of the heart to the left and blunting of the left costophrenic angle. There was diffuse haziness of the left chest from the base to the infraclavicular area, with a heavy left hilar shadow. There was an air-fluid level at the level of the sixth posterior rib on the left. The right lung showed rather marked pulmonary emphysema, heavy fibrotic markings, and an increase in the anterior-posterior diameter especially notable on the lateral visualization. It also showed an area of consolidation extending far to the posterior portion of the chest.

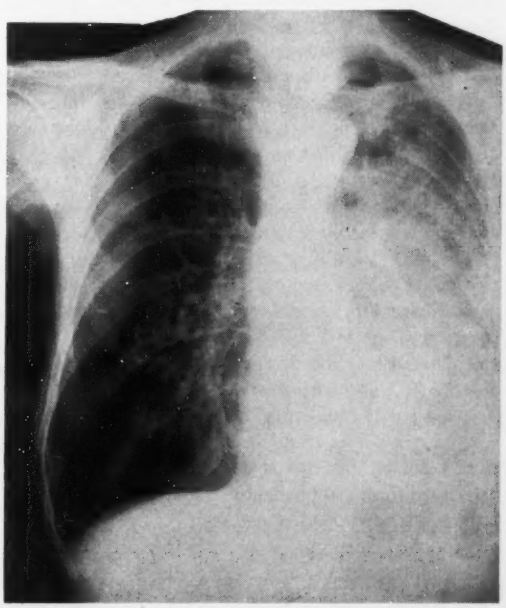


Figure 2. Chest x-ray made November 18.

* Received M.D. degree in June, 1955.

I have the advantage of having seen films taken eight days before these, and I think it makes lots of difference in your thinking. I will read the description of the films taken eight days before. These were outside films that have been returned. "Films taken on November 10, 1954 available for study. There is a rather marked change from this film and the present study. There is a large striking, air-fluid level in the left upper lung field. The lateral films show that this is in the apex of the lower lobe. On this film there is no or little shift of the heart to the left. There is suggestive evidence of a mass posteriorly. There is blunting of the costophrenic angles. There is a suggestion of fluid. Again the pulmonary emphysema is noted. No remarkable infiltration on the right." I think I know what the pathologist found, so I will not go any farther.

Dr. Delp: I want to call on Dr. Fitzpatrick, Dr. Berry, Dr. Weber, and Dr. Christianson later in this case.

DIFFERENTIAL DIAGNOSIS

Mr. David Raab (student)*: The case for today is a 74-year-old white chronically ill male who has a three-year history of bouts of pneumonitis. The first was in 1951; the second was one year prior to admission during which time he had had chronic pulmonary suppuration. He has had symptoms of cough, anorexia, sputum, malaise, weight loss, and other symptoms of a chronic wasting disease.

The physical findings were confined to the chest, including dullness, decreased breath sounds, absent tactile fremitus in the left posterior base, bilateral rales, and bronchial breathing in the right upper lobe.

The signs of pulmonary emphysema were increased A-P diameter and distant heart sounds.

The pertinent laboratory findings were those of compensated respiratory acidosis and a positive smear of acid-fast bacilli reported from another hospital. Sputum culture was positive for *Hemophilus influenzae*. Skin tests were negative. After that time his course was a febrile one resulting in sudden death.

The differential diagnosis today will be based on a productive cough, considering first neoplastic disease. Metastatic lesions could produce a productive cough and could cause the symptoms. This is unlikely because of the chronicity of the disease and the absence of a known primary lesion.

Lymphomas could give this man's symptomatology. The bilateral rales could be due to extensive obstruction. I don't think this is the diagnosis because there was no diffuse lymphadenopathy and no marked bilateral hilar lymphadenopathy.

Bronchial adenoma, a primary tumor, could give this symptomatology and a long history. However, I would like to rule this out because it is more common in young females, and also because he didn't have any conspicuous hemoptysis or wheezing.

Primary bronchogenic carcinoma of the lung could very well be a possibility. It is said that 50 per cent of primary lung carcinomas are first diagnosed as pneumonia. In any pneumonia which does not clear rapidly, carcinoma should be suspected. He is also of the right age and sex. All the symptoms could go along with this except for absence of hemoptysis. He was a chronic smoker, which may or may not add to this diagnosis. I am ruling this out because of the three-year history and because 90 per cent of such cases die within two years. Also we have a negative biopsy and no evidence of hemoptysis.

Next I shall rule out the congenital causes of productive cough, first considering congenital cystic diseases. This man could have an infected congenital cyst or more than one cyst. He could also have a cyst which was harboring carcinoma. I am ruling this out because it is rare, and it would be unusual for this man's symptoms to begin at 70 years from a congenital lesion.

There are infectious causes of productive cough. Regarding the parasitic and fungus lesions, this patient did not have the symptoms, laboratory findings, nor skin tests that would permit this diagnosis.

Tuberculosis is an important possibility. The patient had the cough in the sputum of which was found acid-fast bacilli in another hospital, a sister who died of tuberculosis in her 30's, and he had all the symptoms of a chronic wasting disease. There were lung findings consistent with this disease. He also had clubbing of the fingers. However, I do not think this man had tuberculosis, because of three negative smears in the face of copious amounts of sputum. I feel that we should be able to find acid-fast bacilli at least one time. Also the cultures were negative. Again, this man had two negative skin tests for tuberculosis, and I am sure that this could have been due to anergy. However, I feel that this patient did not have tuberculosis.

The cough, the sputum, malaise, the positive culture of *Hemophilus influenzae,* leukocytosis, fever, and rales all indicate pneumonia. He probably was more susceptible to protracted pneumonia because of his decreased pulmonary reserve secondary to pulmonary emphysema, and because he couldn't cough effectively since the elasticity of his lungs was gone. I feel that this man did have acute pneumonia while in this hospital.

Next I would like to consider chronic lung abscess. The patient could very well have had a lung abscess complicating the pneumonia in 1951, causing a protracted convalescence. Also the chronic cough productive of foul-smelling sputum is found in 90 per

* Received M.D. degree in June, 1955.

cent of patients with lung abscess. The wasting disease with loss of weight, anemia, and cachexia goes along with chronic lung abscess. The circular area in his lung might go along with this, and the fluid level in several areas of his lung would seem to verify this diagnosis. He could have had bronchiectasis secondary to this. I think that this patient did have a lung abscess, or maybe more than one.

The final lesion we ought to consider is bronchiectasis. This man had all the symptoms and signs of bronchiectasis: cough, foul-smelling sputum, weight loss, anemia, and leukocytosis. Some say that *Hemophilus influenzae* is a common organism to culture in cases of bronchiectasis. He could have had pulmonary fibrosis, necrosis, and emphysema secondary to this. Therefore, my final diagnoses are: (1) bronchiectasis with chronic lung abscess, (2) acute pneumonia, while in the hospital, and (3) pulmonary emphysema.

I think that this man probably died of pulmonary infarction, and I think this wouldn't be an unusual finding in a man who has been bedridden and who also has complicating lung disease.

CLINICAL DISCUSSION

Dr. Delp: This patient was found dead in bed. The comment was made that this was not entirely unexpected, but it seems that he died rather suddenly. Can you account for this, Nininger?

Nininger (fourth year student)*: No, it doesn't sound too much like a respiratory death. I think possibly that he could have had a septic embolus to the brain with sudden death. I believe that a pulmonary infarct is possible.

Dr. Delp: Nelson, why was this man disoriented? Was it because of his deafness?

Nelson (fourth year student)*: I think that that accounts largely for why he didn't communicate very well. Outside of that, I can't explain why he should be disoriented. The nonprotein nitrogen wasn't elevated. He had no evidence of neurological signs or symptoms.

Dr. Delp: Dr. Dowel, was this man restless or disturbed during the last day of his life?

Dr. Dowel: No, sir.

Dr. Delp: Schreider, do you have any comments concerning his CO_2 of 36 mEq./L.?

Schreider (fourth year student)*: Well, it's up. It is possible that it could be a complication of bronchial asthma.

Dr. Delp: Does it have any place in this discussion?

Schreider: No, sir.

Dr. Delp: O'Connell.

O'Connell: No, except for the fact that sudden respiratory death might be postulated. He had a very slight pulmonary reserve, and he was probably breathing on his chemoreceptors instead of CO_2 stimulation. These may have eventually failed.

Dr. Delp: Dr. FitzPatrick.

Dr. FitzPatrick: I hope that this statement in your protocol will give you some idea of the appearance of this individual. When Dr. Dowel took me in to see him the night we received him from another hospital, my heart sank. This was a difficult problem to manage as this man had a combined pulmonary insufficiency and an infection. I am sorry that the old x-rays could not be seen by our discussant, as they show without question that this man had an abscess, a rather large one in the superior or apical segment of his middle lobe, and in which a fluid level could be demonstrated.

A few points of interest struck us. Here was a man with an abscess and everything to go with it, who was found in another hospital to have acid-fast bacilli. That is why he was brought here. As the discussant pointed out, if this were a tuberculous abscess there would have been a generous amount of sputum. You would have expected to have found tubercle bacilli or acid-fast bacilli on direct smear of the sputum. This was not found.

The thing that disturbed me most was that we know that most individuals, especially males, have some cause underlying a lung abscess. As Dr. Bachus pointed out in his monograph, the diagnosis of lung abscess in itself is incomplete; it must be lung abscess secondary to something else. Now what was the cause of this man's lung abscess? Most elderly males who are not carefully looked after will have some kind of oral sepsis. Frequently these people aspirate infected material down into the tracheobronchial tree, and thus the lung abscess starts. However, this man was edentulous.

A rule of thumb is that when you have edentulous males with lung abscess you had better start thinking of cancer. We tried to see if this man had a malignancy. We explored the supraclavicular space, and I assure you that the surgeon who did this did it in an atraumatic fashion.

No one was more surprised than I about the sudden death of this man. When we had an opportunity to see why, it was obvious. There are certainly things on his x-rays which we missed and should not have missed.

Dr. Delp: Dr. Weber, I am concerned as to whether the bacillus reported might have been any factor in his illness, and what sort of approach should have been taken with antibiotics.

Dr. Weber: Well, it is reported as the only organism isolated. I think that *Hemophilus influenzae* is an organism that you usually associate with an infection, particularly in children and also in elderly males. I think this did play a part in his infection. The

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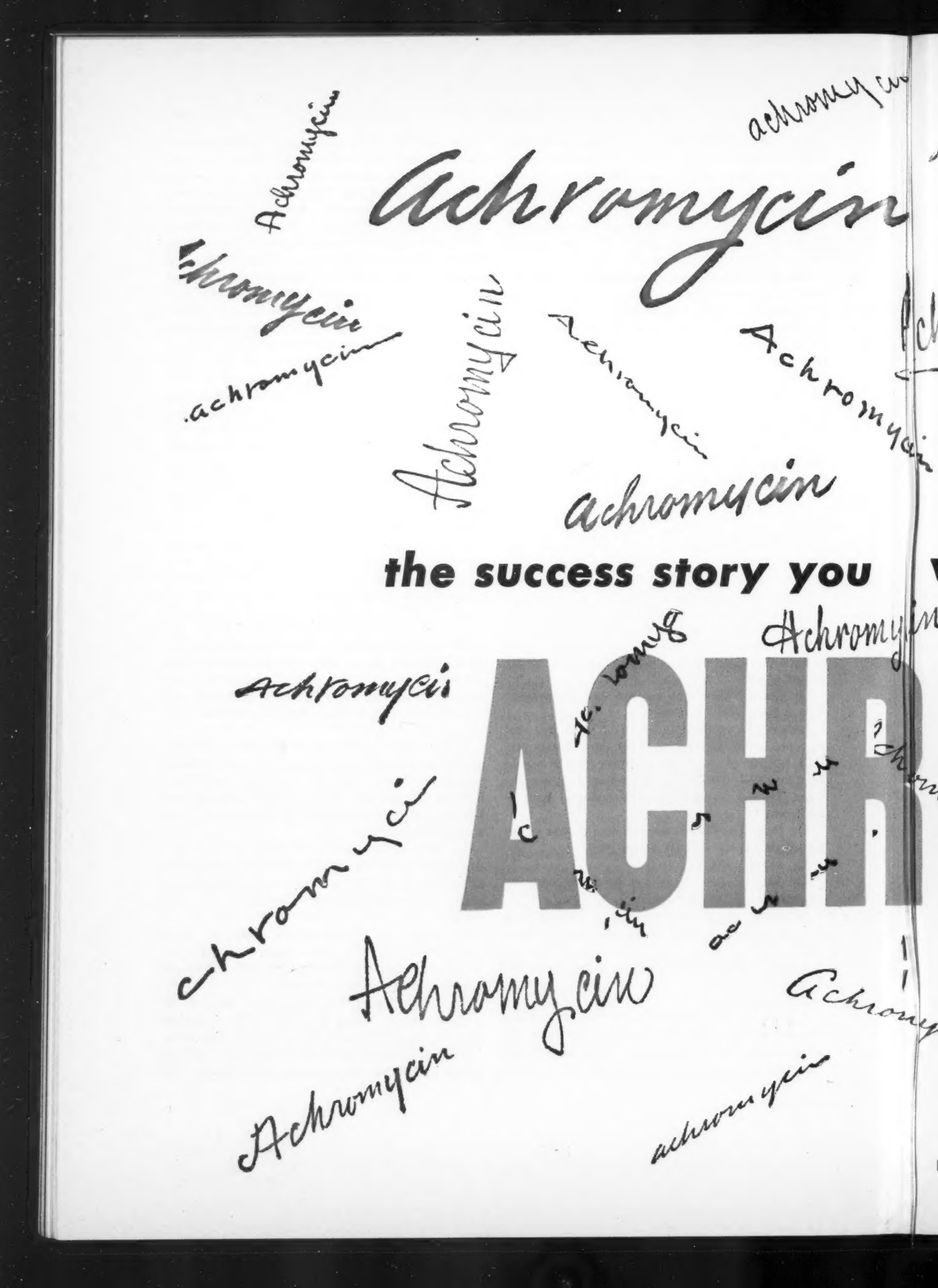
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patient probably did have a lung abscess, as has been shown by x-ray. I think also that the x-ray shows an empyema, and I think he had fluid in his chest. So he had very extensive disease. He had a chronic suppurative disease, and I think he probably had bronchiectasis and lung abscess.

The antibiotic program with *Hemophilus influenzae* is Terramycin and Chloromycetin, but possibly in larger doses than he received.

Dr. Delp: Dr. Berry.

Dr. Berry: Dr. FitzPatrick has made me nervous. I had a diagnosis all lined up until he started talking about different things on the x-ray which we should have seen, and I didn't see anything on the x-rays shown which made me very alarmed about my diagnosis. Now I'm on pretty shaky grounds, but I think from a straight common sense sort of view that this man probably had a lung abscess due to a carcinoma. It is probably an adenocarcinoma of the slow growing type.

Dr. Delp: Dr. Christianson.

Dr. Christianson: Being the last man on the totem pole, Dr. Delp, I think everything has been covered. I was interested a little in the electrocardiogram, but again Dr. Berry and Dr. FitzPatrick shook me up on what I had missed on the x-ray. I thought that with the absence of R waves the patient might have an old infarct on the basis of the electrocardiogram which I have seen, although with the low voltage he well may have had another one.

I think the student's comment about lung abscess is well taken. In my brief experience I think that there are findings of brain abscess. I think that brain abscess is a handmaiden of lung abscess.

Dr. Delp: No one has mentioned unresolved pneumonia. Why not? Isn't it popular to make this diagnosis any more?

Dr. FitzPatrick: I was told never to use the term.

Dr. Berry: I have learned never to use it.

Dr. Lin: Dr. Delp, there are a few words I would like to say. I read the protocol only a few minutes ago, and there are no indications that this patient had a constrictive pericarditis. I think that this electrocardiogram is from an old man with coronary ischemia. I doubt seriously that this man had evidence of myocardial infarction because the chest leads are not too far from normal. His electrocardiogram low voltage is due probably to ischemia and poor nutrition.

Dr. Delp: Everybody is studiously avoiding the CO_2 although I have tried to get some comment. Dr. Bolinger, it has been mentioned that this patient had a compensated respiratory acidosis. I want to know whether his CO_2 at this level might have contributed in any way to his death. If so, is this something that could have been avoided?

Dr. Bolinger: Well, with the finding of respiratory acidosis, and I think that this is what it was, we know that sudden death is not uncommon at this age. We also know that this patient had a large fluid-filled cavity. With a borderline respiratory reserve, I think it is possible that he might have turned over in bed and drowned all at once. Fluid from this cavity rupturing and pouring down another bronchus or something of that sort would have decreased his respiratory reserve so that he died.

Dr. Delp: How could this CO_2 have been altered favorably?

Dr. Bolinger: The only possibility of compensating respiratory acidosis is by increasing respiration. This might have been done with a respirator, or more practically by getting rid of his infection.

Dr. Lin: The darkness of his blood was mentioned, and I think that is a very good point because in chronic chest disease you often see a severe anoxia, and the patient won't recover because of a complicating infection. If you do oxygen studies in this type of patient, you will find severe anoxia. This patient may have had a very severe anoxia which would cause an arrhythmia of the heart.

Dr. Delp: I think those are pertinent comments, Dr. Lin.

PATHOLOGY REPORT

Dr. Peters (pathology resident): The autopsy on this patient was done 10 hours after death. External examination revealed an elderly man whose weight was approximately 120 pounds, and who was markedly emaciated. There was a recent incision in the left supraclavicular area and an ulceration in the left cheek close to the nose. This had a brown discoloration, which we judged to be a skin carcinoma of the face.

When the body was opened, we were struck immediately with the intense foul odor which was reported during his clinical course. On dissecting the chest we found, on the left, marked fibrinous adhesions over the pleura, particularly over the left lower lobe and the lower part of the upper lobe. While dissecting farther we broke into a large empyema cavity, which contained approximately 1200 cc. of greenish yellow fluid which was blood tinged. Cultures taken from this fluid were reported as negative.

The lungs were heavy and firm, weighing approximately 2050 grams together, and on cut section many hemorrhagic elevated nodules were recognized in almost all lung fields. They were from 1 to 2 centimeters in diameter with some larger and some smaller. The bronchi were filled with purulent mucus, and the mucosa of the bronchi was blood streaked and eroded. Dissection of the bronchi into this large

empyema cavity showed that they were slightly dilated, but not bronchiectatic. None of the bronchi which were traced out led to the area of the empyema.

The chest had the contour of emphysema. The upper lobes were markedly emphysematous. The hilar lymph nodes were markedly enlarged and, as reported on the x-ray, there were calcified nodules along the left lower lobe.

The liver was firm, weighing approximately 1230 grams, and slightly congested. The spleen was also congested. The kidneys were in almost perfect shape for a man of this age, though there were a few small retention cysts. The heart weighed 300 grams and was almost in too good a condition for a person of this age with this kind of disease. There was a slight indication of an acute mild fibrinous pericarditis. This could be seen only microscopically and not grossly. The valves also showed slight change. There was mild indication of verrucous endocardiosis. However, microscopically there was no real endocarditis.

Cross section of the brain showed a large cystic space of approximately 1.5 cm. in diameter in the left cerebral hemisphere in an area ranging from the anterior portion of the caudate nucleus to the left of the optic chiasm, lateral and superior to the thalamic area. This was lined by a yellowish wall. The cyst itself was filled with clear yellowish fluid.

I think Dr. Wahl will talk about the microscopic pathology of the lungs. The liver showed marked congestive changes. In such areas there was mild evidence of cardiac cirrhosis. The cyst in the brain was an old cyst lined by macrophages which extensively took up iron as shown on the Perle's stain. There were also many foamy macrophages and chronic inflammatory reaction. The bone marrow was slightly hyperplastic, particularly where the white series was concerned.

Just a few words on the lungs. We did serial sections on the lungs in numerous areas and we did all sorts of staining procedures. We did not find any acid-fast bacteria in any of these areas. Many gram positive cocci were found both in the fresh and in older areas. The bacterial studies of the lungs done at the time of the autopsy showed non-hemolytic staph aureus, green streptococcus, and aerogenes. As I have said before, the empyema cavity was sterile.

Dr. Wahl: There are three lesions of special comment. One is in the brain. The brain lesion of course is an old hemorrhage, at least of some months' duration. It had a hemorrhagic wall.

The other two lesions had to do with the lungs. The most striking thing present was the enormous empyema. In this connection it should be remembered that the empyema cavity was bacteriologically negative. The microscopic sections showed very little evidence of acute inflammatory reaction. It was essentially chronic.

A section of the liver showed considerable passive congestion. The severe passive congestion would suggest a failing heart. This had perhaps developed in the last three days.

There were numerous small abscesses in the lung. This was also a pneumonic area.

A section of the lung showing an abscess also showed a curious body that somewhat resembled actinomycosis. These peculiar staining bodies are not typical of actinomycosis, but they suggest some type of mycotic infection. A very delicate network of mycelia running criss-cross and in various directions is an interesting thing to find in the midst of this purulent material.

Dr. FitzPatrick: In regard to the question you asked Dr. Bolinger, in this particular elderly male individual it would have been very difficult to increase his ventilation—almost impossible. We might have tried to give him some positive pressure breathing with room air plus oxygen in an attempt to wash

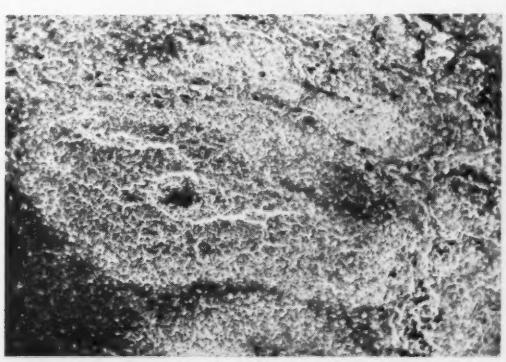

Figure 3. Photomicrograph of lung, showing area of bronchopneumonia with early abscess formation. Note clumps of organisms resembling a colony of actinomyces.

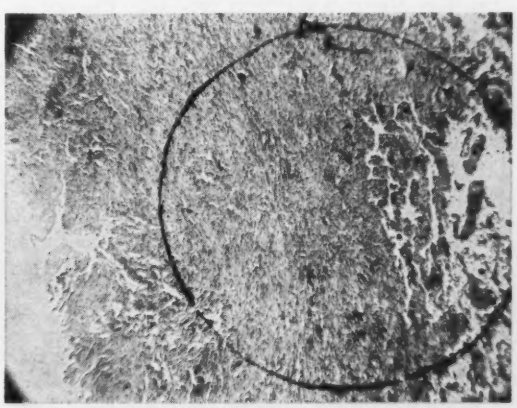

Figure 4. Section through wall of old empyema cavity. Note its heavy fibrous character and the paucity of inflammatory cells.

out CO_2. I think that there is a chance that if we had tapped his chest to remove the empyema, that might have been effective.

Dr. Delp: What do you think about his illness from 1953 on? Where along the line might this outcome have been changed?

Dr. FitzPatrick: Well, this fund of information is all here this morning, but at the time the patient was in here, it was hard to get from scattered members of his family. Actually his living quarters were very poor, and it was difficult to determine when he had the onset. It is possible that he might have had a lung abscess for a whole year.

Dr. Delp: Do you know what happened on the seventh day to cause his fever, Dr. FitzPatrick?

Dr. FitzPatrick: No, sir, I don't. I presume he had a local extension of the infection in his left chest. Possibly the empyema spread farther, but I would be guessing at that.

Dr. Delp: How long had this man been hospitalized before he came here?

Answer: One week.

Dr. Delp: Dr. Weber, is there any time within that one week that other antibiotic therapy might have salvaged this patient? Or do you have that much faith in the ability to cure with antibiotics?

Dr. Weber: No. Not unless it was drained at that time.

Dr. FitzPatrick: I think from the fact that he had an abscess with lots of purulent sputum from which the only organism our laboratory could isolate was *Hemophilus influenzae,* we felt quite sure that he had been receiving a goodly amount of penicillin in the other hospital and probably on the outside. I was not too impressed with the fact, looking on him that first week, that he was having a virulent infection of his lungs due to *Hemophilus influenzae.* I thought it was more or less there as a contaminating organism. His flora had been previously changed by all the other chemotherapy I presume he had received.

Dr. Orr: Well, of course it is foolish to think that you can cure the empyema without surgery. You can't cure abscess anywhere without drainage. Now someone will point his finger at me and say that an appendix abscess will resolve, but what they don't know is that perforation into the gut is the method of resolution. In such a case, drainage has been established. I suppose a small abscess in the tip of one's finger will resolve, but a real abscess that has been walled off requires drainage.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Primary

Acute and chronic bronchopneumonia of all lobes of the lung, advanced, with two bronchopleural fistulae in the left lower lobe (history of long-standing productive cough).

Possible lung abscess of left lower lobe with rupture.

Empyema of the left chest, 1200 ml.

Fibrous adhesions between the parietal and visceral pleura over the lower lobe of the left lung, advanced.

Fibrinous pericarditis, slight.

Acute tracheitis, moderate.

Hyperplasia of all tracheobronchial lymph nodes, moderate.

Hyperplasia of the hemopoietic system of the bone marrow, slight.

Emphysema of both upper lobes and right middle lobe of the lungs, moderate.

Dilatation of the right side of the heart, moderate.

Acute and chronic passive congestion and cardiac cirrhosis of the liver, moderate.

Fatty metamorphosis of the liver, moderate (history of 25-pound weight loss over period of one year and of inadequate diet).

Submucosal hemorrhages of the stomach, duodenum, and focally within the small intestine and rectum, moderate.

Small erosions within the duodenal mucosa, slight.

Varicose veins in the lesser curvature of the stomach, slight with dilatation of veins in the esophagus.

Focal petechial hemorrhages over the mitral valve of the heart, slight.

Recent linear incision over the left clavicle (history of lymph node biopsy one day before death).

Emaciation, body length approximately 70 inches, weight approximately 120 pounds. Tubular deformation and lipid depletion with focal areas of intertubular hemorrhages of the adrenal, moderate.

Vacuolization of the basophilic cells of the pituitary, slight.

Multiple puncture wounds in both antecubital fossae, both forearms and upper arms (history of venoclyses and injections for 12 days before death).

Accessory

Verrucous endocardiosus of the mitral and tricuspid valves, slight.

Fusion of the leaflets of the aortic valve, slight.

Fenestration of the posterior leaflet of the aortic valve.

Arteriosclerosis of the ascending aorta, of the basilar arteries of the brain, of the coronary arteries, slight; of the lower part of the abdominal aorta with ulceration and thrombus formation, moderate.

Remote infarct with cystic degeneration within the left cerebral hemisphere.

Calcified nodules in the left pulmonary hilar region.

Multiple small fibrous nodes within liver and spleen.

Nodular hyperplasia of the prostate, slight (history of nocturia and frequency for unknown duration).

Atrophy of both testes, moderate.

Hyalinization of islets of the pancreas, slight.

Osteoporosis of the vertebral column, slight.

Hypertrophic arthritis of both knee joints, moderate.

Mycosis of the big toe of the right foot, advanced.

Possible basal cell carcinoma of the skin over the left nose and cheek.

Kansas State Pediatric Society to Meet

A scientific program will be presented by the Kansas State Pediatric Society at a meeting to be held at the Broadview Hotel, Emporia, on Saturday, September 10. Dr. James Baty, Boston, and Dr. Herbert A. Wenner, of the University of Kansas Medical Center, will be speakers.

Dr. Baty will present three papers on the following subjects: "Psychosomatic Problems," "Emergencies in the Newborn," and "Colic." Dr. Wenner will address the group on "Current Research in Poliomyelitis."

The day's program will begin at 10:00 in the morning. Two papers will be presented before the luncheon meeting and two papers in the afternoon, followed by a business meeting and dinner.

Registration is not limited to pediatricians. All physicians who are members of the Kansas Medical Society are eligible to attend.

Board of Health Appointments Made

Dr. Ivan W. Cain, Kansas City, Dr. Richard E. Speirs, Dodge City, and Mr. D. C. Wesche, Manhattan, were recently appointed members of the Kansas State Board of Health by Governor Fred Hall. Their terms will expire in May of 1958.

The terms of four members of the board will expire in May of 1957. Those four are Dr. H. St. Clair O'Donnell, Ellsworth, president; Mr. Wilber Murray, Hutchinson, vice-president; Dr. H. Penfield Jones, Lawrence, and Dr. George M. Coffey. Dr. Coffey is a dentist.

Remaining members of the board, whose terms will expire in May of 1956, are Dr. Robert C. Polson, Great Bend, Sr. M. Aloysia, Wichita, and Dr. Thomas P. Crispell, Parsons. Dr. Crispell is a veterinarian.

Television Programs for 1955

Twenty-five television programs planned for local showings are being prepared by the A.M.A. Bureau of Health Education and are now available. Medical societies may arrange for showings.

One series, "What To Do," consists of six five-minute films on backache, hay fever, eye injury, skin problems, baseball finger, and dizziness. "Script clips" include six films and accompanying scripts to be narrated by a local doctor. The subjects are normal eyesight and common defects, exercise and your heart, industrial accidents, the nervous system, polio, and prevention of crippling in arthritis.

A series of 13 rural health scripts, to be used in live participation shows, covers rabies, brucellosis, home pasteurization of milk, pure water supply from farm wells, balanced diet, septic tanks, the place of minerals and vitamins in diet, food for growth and medical care, weight control, health examinations, family physician, accidents in the home, and health insurance.

Polio Research Continues

The search for strains of live virus suitable for use in polio vaccine will be continued at the University of Kansas Medical Center under a grant of $105,116 from the National Foundation for Infantile Paralysis. The grant was awarded on July 1.

Dr. Herbert A. Wenner, research professor of pediatrics, will direct the research, assisted by Dr. C. Arden Miller, assistant professor of pediatrics; Dr. Jacqueline Baumeister, instructor in pediatrics, and Dr. George Dubes, research associate.

Golden Belt Society Meets

Members of the Riley County Medical Society were hosts to the Golden Belt Medical Society at a meeting held at the Manhattan Country Club on July 14. The afternoon program consisted of a panel discussion on hypertension with Dr. E. Gray Dimond, of the University of Kansas Medical Center, as moderator. Participants were Dr. Severt A. Anderson, Clay Center; Dr. Robert M. Carr, Junction City; Dr. Alfred J. Horejsi, Ellsworth, and Dr. Robert Sohlberg, McPherson. An informal program followed the dinner hour.

"The doctor draft is unjust. Its extension would mark the first time in our history that any group of citizens has been singled out for conscription in peace time because of their professional skill."—*Chicago Tribune, June 10, 1955.*

Infectious Hepatitis Complicating Pregnancy

Robert H. Finkle, M.D.

Kansas City, Missouri

The metamorphosis of acute yellow atrophy of pregnancy into pregnancy complicated by infectious hepatitis has indeed been slow. In 1937, the American Committee of Maternal Welfare classified acute yellow atrophy of the liver as a toxemia of pregnancy, and this same classification persisted up to 1947.[1] During this period of time, many authors believed that liver damage was a possible etiologic agent of toxemia of pregnancy. This viewpoint was inferred from the association of hypertension, albuminuria, edema, and convulsions in patients terminating with acute yellow atrophy of pregnancy.

However, in 1947 Zondek[2] showed that infectious hepatitis complicating pregnancy could assume three different clinical courses and that it was in the most severe form of the disease that acute yellow atrophy occurred. Subsequent to this publication, enthusiastic support of this opinion was shown by Mallory,[3] Wood,[4] Lucke,[5] Dill,[6] and others, so that today acute yellow atrophy is no longer thought of as a disease peculiar to pregnancy, but rather that it is the terminal phase of severe liver damage. The etiology of this damage is of an infectious or poisonous nature. It is with the former, specifically viral infectious hepatitis complicating pregnancy, that this paper is concerned.

The specific etiologic agent of infectious hepatitis is a filterable and heat stable virus, as is the agent of homologous serum jaundice.[7, 8, 9] Homologous serum jaundice implies that transmission of the disease is through the medium of infected plasma, serum, whole blood, and contaminated needles and syringes. The mode of transmission of infectious hepatitis is by direct or indirect contact with feces of persons suffering from the disease, although nasopharyngeal washings have been shown to transmit infection in human volunteers, as have contaminated needles and syringes.[8, 9, 10, 28]

The incubation period of infectious hepatitis is 8 to 42 days with the disease having its appearance in about 30 days following contact with contaminated material in the majority of cases.[7, 11, 12] The incubation period of homologous serum jaundice varies from 40 to 180 days with an average of 55 to 135 days following inoculation.[8, 9] No cross immunity between serum hepatitis and infectious hepatitis has been demonstrated.

This lack of cross immunity, the different incubation periods, the different modes of transmission, the failure of homologous serum jaundice to spread from patient to patient, and frequent spread of infectious hepatitis are the best evidence that homologous serum jaundice and infectious hepatitis are two distinct clinical entities, either as two entirely different agents or two immunologically distinct strains of the same agent.[8, 9]

The pathologic lesions of infectious hepatitis and homologous serum jaundice are essentially the same.[13] There is an acute zonal hepatitis with centrilobular necrosis and autolysis of parenchymal cells, disorganization of the liver cell columns, and a leukocytic and histiocytic infiltration of the periportal areas.[9, 13] The reticular framework of the liver lobule is not destroyed, and regeneration of surviving parenchymal cells results in restoration of the normal lobular pattern. This is the usual sequence of events with recovery being the rule.

Massive hepatitis with acute liver atrophy may ensue, however, when there is an excessive virulence of the infective agent, or when there is an increased susceptibility to the agent of ordinary virulence.[13] Such susceptibility is a consequence of previous liver damage, exposure to hepatotoxic agents, malnutrition, alcoholism, and pregnancy. This, as will be seen later in the discussion of the clinical course of the disease, is the important factor in the management of maternal hepatitis; the prognosis of the patient depends primarily upon the extent of the hepatic lesion. If the centrilobular necrosis is not severe, the prognosis is excellent. However, if the hepatitis proceeds to the extent of generalized massive hepatitis, subacute massive hepatitis with post-necrotic scarring or death from acute liver atrophy is the result.[13] The latter is the more common course of events when massive hepatitis is associated with pregnancy.

Infectious hepatitis complicating pregnancy is not unlike infectious hepatitis in the non-pregnant individual in that it may exhibit a marked variation in severity of clinical symptoms and laboratory findings. Clinically there are four stages of the disease: pre-icteric, icteric, convalescent, and the final or end stage of the disease.

In the pre-icteric stage, symptomatology consists of anorexia, nausea, vomiting, asthenia, vague epigastric

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This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Finkle is now completing his internship at St. Mary's Hospital, Kansas City, Missouri.

distress, and constipation. These symptoms are not specific and are easily misinterpreted as common phenomena of pregnancy. Physical findings may be lacking, although there may be right upper quadrant tenderness, low grade fever, lymphadenopathy, and palpable liver enlargement. Hepatic enlargement frequently occurs prior to the onset of jaundice.[9, 13]

The icteric stage is marked by the onset of jaundice. Gastrointestinal complaints persist and may be accentuated by the onset of intractable vomiting. Headache, restlessness, and psychic instability may occur. Physical findings include increased liver enlargement to 3 to 4 cms. below the costal margin, splenic enlargement in 85 per cent of cases, and bradycardia. Hepatomegaly usually correlates with the prognosis of the disease, reaching a maximum on the average in 16 to 17 days and then starting to recede.[9]

The convalescent stage is marked by a recession of clinical and laboratory findings. This follows the icteric stage and is the usual sequence of events. However, progression to the final or end stage of the disease may occur. This latter is marked by an increased icterus, diarrhea, mental confusion and apathy, tremors, flapping movements of the hands and wrists, muscular rigidity, abnormal reflexes, dilated pupils, rapid and thready pulse, fetor hepaticus, hemorrhagic phenomena, and decreased liver size. These are late and ominous signs indicative of acute liver atrophy and hepatic failure.

Laboratory findings in the pre-icteric stage may be lacking. Swift et al.[27] recorded bilirubinuria and an increased urobilinogenuria as the first detectable abnormal laboratory findings. Sodeman[9] noted elevated thymol turbidity and cephalin flocculation reaction as well as abnormal bromsulfalein and alkaline phosphatase retention in approximately one-half of his cases. The icterus index and van den Bergh tests are elevated prior to the onset of jaundice. The van den Bergh is at first indirectly, and later both directly and indirectly, elevated with further increments of the icterus index. Other findings of the icteric phase include leukopenia, depressed total protein, especially the albumin fraction with increased globulin fraction, prolonged prothrombin time, abnormal bromsulfalein and alkaline phosphatase retention, and further elevations of the thymol turbidity and cephalin flocculation reaction.

These findings become reversible as the patient enters the convalescent stage, although persistence of an elevated thymol turbidity and cephalin flocculation reaction for one to three months is not unusual. Progressively decreasing blood urea level with elevated amino acid and non-protein nitrogen, decreasing total cholesterol and cholesterol ester blood level, absence of urinary bile, decreasing total protein with inversion of the albumin-globulin ratio, elevated serum ammonia, elevated lactic and pyruvic acid levels, and persistent hypoglycemia in the presence of excessive carbohydrate intake are signs of severe impairment of liver function. They signify the end stage of the disease with acute liver failure.

The differential diagnosis of jaundice in the pregnant and non-pregnant individual is identical with one exception—toxemia of pregnancy may produce jaundice. Biliary tract disease, chemical intoxication, neoplasia, heart failure, and hematologic, infectious, and parasitic processes must be differentiated.

Biliary tract obstruction and cholecystitis are common, while maternal infectious hepatitis is rare. X-ray examination will reveal stones and a non-functioning gallbladder, while increased alkaline phosphatase and bilirubin in the presence of decreased or absent fecal and urinary urobilinogen will indicate obstruction. The lack of a history of exposure to chemical poisons and other toxins, including mushrooms (Amanita phalloides) and snake venom, rules out the possibility of chemical poisoning. Cirrhosis is differentiated by a history of alcohol ingestion and poor nutrition with physical findings of portal hypertension—ascites, splenomegaly and esophageal varices, spider angiomata, liver palms, and liver punch biopsy.

Neoplastic disease, primary or metastatic, may require in addition to a careful history and physical examination the use of x-ray examination, the laboratory, and lymph node biopsy. Although malignancy is less frequently seen in the child bearing age, leukemia, Hodgkin's disease, secondary liver metastases, and primary carcinoma of the head of the pancreas, liver, and bile ducts must be considered. Heart failure is differentiated by physical findings alone. Patients with homologous serum jaundice will present a history of exposure and a more prolonged incubation period.

Transfusion reaction must be considered in patients receiving whole blood transfusions. Sickle cell anemia, less commonly found in colored females than in the male, will present sickling of the erythrocytes and an increased erythrocyte fragility. Splenic anemia exhibits splenomegaly, characteristic blood cell findings, and an increased indirect with a normal or slightly elevated direct serum bilirubin. Infectious processes such as typhoid and typhus fever, Weil's disease, syphilis, tuberculous hepatitis and cholangitis, and parasitic infestations such as Echinococcus, amebiasis, yellow fever, and malaria may be distinguished by a history of exposure and characteristic clinical and laboratory findings.

The greatest difficulty in diagnosis occurs in the pre-icteric stage of the disease when the symptom complex of anorexia, nausea, vomiting, asthenia, and epigastric distress is interpreted as common phenomena of pregnancy. Frequently the patient's complaint will be that of a recent upper respiratory infection or a persistent attack of the "flu." Physical examination

and laboratory findings at this time may be entirely negative, and the patient may be dismissed without arrival at the correct diagnosis. A history of exposure to hepatitis or a recent outbreak of epidemic hepatitis increases the likelihood of the disease.

The interval of time between the onset of symptoms and the appearance of jaundice is 2 to 18 days with an average of 5 to 14 days.[9] Corresponding liver enlargement and tenderness, lymphadenopathy, and suggestive laboratory findings may be present at this time to confirm the diagnosis. A high index of suspicion followed by a repeat examination in 6 to 10 days will make earlier diagnosis possible and thereby greatly improve the prognosis.

Infectious hepatitis in the first and second trimesters of pregnancy seems to carry no more serious a prognosis for the mother than infectious hepatitis in the non-pregnant individual; patient recovery depends upon severity of the hepatitis. However, once the patient enters the third trimester and approaches term, the maternal mortality is adversely affected and fetal salvage is reduced. Pregnancy, during the last trimester especially, is therefore a decisive factor in aggravating the clinical course of infectious hepatitis. Why the last trimester of pregnancy has such an effect is not definitely known. However, it is known that the severity of infectious hepatitis is increased in cases of undernutrition.[13]

Evidence is available that nutritional deficiency is a factor tending to promote or at least favor liver injury in man and laboratory animals.[13] In the patient with an excessive amount of vomiting during the first and second trimesters, and the resultant decreased dietary intake secondary to nausea and anorexia, nutritional reserves are decreased and may even be depleted. Add to this the increased need of dietary protein during pregnancy, and it is easy to speculate that undernutrition is an important factor in the conversion of simple hepatitis of pregnancy into that of acute yellow atrophy. Then, too, impairment of the portal circulation may result secondarily from an enlarged uterus impinging upon the portal blood vessels. Therefore it is to be remembered that the prognosis may be poor in the third trimester and grave when undernutrition is concomitantly present. Also, as is the case in any other disease process, early diagnosis and institution of therapy will improve the prognosis. If the patient is moribund or comatose before therapy is instituted, little is to be expected from the best means of management.

Fetal and neonatal prognosis in maternal hepatitis varies with the severity of the clinical course of the disease. When mild or moderately severe hepatitis is encountered, pregnancy will, in all probability, follow its normal course. However, in severe hepatitis premature delivery and spontaneous abortion may be expected, depending upon the trimester of pregnancy. This abortive action results from the influence of the liver failure or from the debilitating effect of the disease upon the pregnant mother. It has been shown experimentally that liver damage and failure can induce abortion.[26] The extirpation of one-third of the liver does not prevent the normal course of pregnancy, whereas the excision of one-half or more of the liver results in the immediate termination of pregnancy.[26]

Gellis et al.,[36] Stokes and Neefe,[37] and Havens[38] have demonstrated the value of gamma globulin administered parenterally in the prevention of infectious hepatitis. This is of value in preventing the disease if given prior to the onset of symptoms, but is of no value in prevention once symptoms have occurred. Recommended doses vary from 0.06 to 0.15 cc. per pound of body weight given intramuscularly. Passive immunity continues for six to eight weeks.[9] The immediate use of prophylactic gamma globulin in patients exposed to hepatitis or during outbreaks of epidemic hepatitis is of definite value and is to be encouraged.

In the successful management of maternal infectious hepatitis, one must bear in mind the dual nature of the problem that exists. Not only must active therapy be instituted promptly to prevent further and more severe liver damage, but special attention must also be devoted to the course of delivery and postpartum care. However, due to the primary nature of the disease and the associated high mortality rate, management of hepatitis is of prime importance. It is necessary, as a result of the infectious nature of the disease, to isolate the patient and to use isolation technique with the patient's excreta and with contaminated needles and syringes.

The liver has remarkable regenerative power and exhibits spontaneous reproduction of functioning liver parenchyma if the action of the injurious agent is arrested and the necessary dietary essentials are available for cellular reproduction. Indeed, as long as the reticular framework of the lobule is intact and there are surviving parenchymal cells, regeneration will result in restoration of the normal lobular pattern.[13] Since there is at this time no specific anti-viral agent available, management must be directed along the course of decreasing liver susceptibility to damage and promotion of optimal conditions for regeneration of the liver parenchyma. To fulfill these needs, complete bed rest, diet, vitamin supplements, and the avoidance of additional trauma to the liver are mandatory.

Factors injurious to the liver that are to be avoided include surgical operations, secondary infections, and the exposure to hepatotoxic drugs. Operative procedures are contraindicated because of tissue trauma incident to surgery and the use of anesthetics, which are hepatotoxic. Ether, chloroform and ethylene are

the most dangerous anesthetics in liver injury.[32] Secondary infections of all types produce adverse effects in infectious hepatitis and therefore require prompt attention. Alcohol, opiates, and barbiturates are contraindicated in all stages of hepatitis. Alcohol produces a toxic effect, whereas the latter drugs produce prolonged and exaggerated effects and are capable of precipitating hepatic coma.

The utilization of a high carbohydrate, high protein, low fat diet is indicated in infectious hepatitis.[9, 13, 25] Opie[14] and Mann[15] have shown that high carbohydrate intake prolonged the life of experimental animals with induced liver damage. Mann and Bollman, working with animals poisoned with phosphorus and carbon tetrachloride respectively, showed that the animals maintained on a high carbohydrate diet lived longer than the animals not maintained on such a diet, other factors being equal.[24] Mann, working with Eck fistula dogs with complete hepatectomy, showed that the dogs died from the effects of hypoglycemia long before signs of liver insufficiency could supervene unless large quantities of glucose were administered.[23] In addition to the above, glucose is necessary for glycogen metabolism of the liver and for its conjugation of phenol groups with glucuronic acid.[16] Therefore, the indications for a high carbohydrate diet are threefold: to prevent further liver damage due to nutritional deficit, to prevent death secondary to hypoglycemia, and for the protein sparing action of carbohydrate.

Patek reported in 1937[17] and later in 1948[18] that a high protein intake was indicated in liver injury. Definite liver lesions have been extensively reported as occurring in animals maintained on diets with insufficient protein.[13, 19, 20, 21] Suffice it to say, without quoting the extensive literature on this subject, that a high dietary protein is definitely indicated in infectious hepatitis. The value of a low fat diet stems from the fact that it is believed that diets rich in fats provide an increased susceptibility to liver damage by predisposing to fatty infiltration of the liver.[13, 22] However, not all authors believe that fat restriction is necessary in the presence of adequate dietary protein.

At this center, dietary management consists of a high protein, high carbohydrate diet without fat restriction—specifically the Patek diet. This consists of 140 grams protein, 375 grams carbohydrate, and 50 to 100 grams fat. This total intake is dispersed throughout the day with interval feedings. Supplementary vitamin therapy with the B complex group (thiamine, riboflavin, nicotinamide, pyridoxine, and pantothenic acid) and vitamins A, C, and K has been recommended, especially if the dose is therapeutic without large unbalanced doses of individual vitamins. Vitamin B_{12} has been recommended as it stimulates protein regeneration and exerts a lipotropic action. The value of methionine and choline is suggestive but not proved, and its use depends upon the individual physician.[32]

In severe liver damage with the appearance of irrationality, irritability, and unresponsiveness of the patient signifying impending hepatic coma, therapy becomes more difficult. The patient is no longer capable of taking sufficient nutrition orally, and this must therefore be given via stomach tube or intravenous infusions or both. The former is preferable but less possible without the patient's co-operation. Intravenous infusions of dextrose, protein hydrolysates, and amino acid solutions are the best means of supplying the patient's nutritional requirements. Large amounts of dextrose up to 3 to 4 liters daily may be necessary to prevent hypoglycemia.[41, 42] Whole blood and albumin solutions are indicated to correct secondary anemia and depressed serum proteins.

With the low dietary intake and diarrhea associated with hepatic coma, there is a depression of the serum electrolytes, specifically sodium, potassium, magnesium, and calcium.[44] A metabolic acidosis occurs secondary to elevated ketoglutaric, pyruvic, and lactic acids. This is a consequence of the inability of the liver to remove these acids.[44] Therefore, correction of electrolyte disturbances and altered acid-base balance remains a constant problem. Oliguria, frequently associated with hepatic insufficiency and coma, results in an increased circulating blood volume. Caution must therefore be exercised in vigorous intravenous therapy to prevent the development of cardiac decompensation.

Impairment of the ability of the liver to convert ammonia into urea results in an elevated serum ammonia. This produces cellular intoxication and aggravates the clinical picture of hepatic insufficiency. The production of ammonium intoxication presents a picture resembling that seen in hepatic coma.[44, 45, 46] Ammonium salts are therefore contraindicated. The administration of glutamic acid decreases the serum ammonia level. Such action results from the utilization of ammonia in the conversion of glutamic acid to glutamine.[41] Antibiotics and non-absorbable sulfa preparations that inhibit bacterial growth in the intestinal tract and thereby rid the tract of urea-splitting organisms are beneficial. What urea is produced is then excreted, whereas previously some of the urea would have been converted back into ammonia. This may ultimately be the explanation of the unknown beneficial effect of Aureomycin in the treatment of hepatic coma.[59]

The use of ACTH and cortisone is controversial. They have been shown to produce marked improvement in severe cases of hepatitis. However, there are certain dangers associated with their use in maternal hepatitis. These are sodium and water retention and potassium depletion, which further aggravate the dis-

turbed electrolyte imbalance. Oliguria and diarrhea associated with hepatic coma contraindicate their use. Then, too, the dose of cortisone must be reduced gradually following active therapy. Otherwise the patient will regress to a clinical status worse than that prior to its use. Cortisone is of value in selected cases if it is used properly to prevent adrenal insufficiency secondary to its withdrawal.

Sedation is necessary to control irritability and convulsions. Morphine and barbiturates are contraindicated because of prolonged action and effect as mentioned previously. For control of excitability, demerol or codeine and aspirin is satisfactory. In the patient with extreme irritability, excitability, and convulsions, paraldehyde is the drug of choice.

In the obstetrical management of maternal hepatitis, the objective is to bring the patient to term with delivery of a viable fetus while keeping maternal mortality at a minimum. During the first and second trimesters the severity of the disease is mild with resolution of the disease following bed rest, adequate nutrition, and general supportive therapy. Obstetrical interest is that of any other infectious process complicating pregnancy, that is, to correct any abnormal disease process which may produce complications when the patient nears term. The difficulty in obstetrical management arises when the patient in the late second or early third trimester with a previable fetus develops a severe hepatitis with impending hepatic failure. Should the patient be allowed to go to term when pregnancy is known to exert a deleterious effect upon maternal hepatitis, or should artificial interruption of pregnancy be performed when the procedure itself is capable of inducing further liver damage and precipitating hepatic failure?

If there are positive signs that the fetus is dead, the course is obvious. It is that of waiting for spontaneous delivery of the fetus, while active and vigorous therapy is directed toward reducing the severity of the hepatitis. Artificial interruption is not to be accomplished as this offers the patient nothing other than exposing her to further liver damage from the procedure itself. The patient will abort spontaneously at a later more favorable date, and there are no harmful effects resulting from the retention of the uterine contents.

With a previable fetus, every effort should be made to carry the patient to term with a normal spontaneous delivery. The delivery should be accomplished by the simplest, easiest, and least disturbing method to the maternal physiology. However, if acute hepatic failure appears inevitable, artificial interruption of the pregnancy seems justified by the fact that death of the mother may ensue or the product of gestation may be a dead, macerated fetus if the pregnancy is allowed to continue. If interruption is planned, it should be performed long before the hepatitis has become severe, for such a procedure is itself capable of precipitating hepatic failure and death.

The earliest time for such a procedure is limited only by the physician's capability and facilities for the care of premature infants without increased mortality. Since the early prediction of hepatic failure is not absolute, it is easy to see that the fetus will be exposed unnecessarily to the hazards of prematurity in cases that may have had a benign course. If one waits to predict hepatic failure with certainty, it is then too late to carry out interruptive procedures without danger to the mother. The other possible course is that of expectant waiting to see if the patient will improve without developing massive hepatitis. In this way, the fetus is not endangered by prematurity and there is no risk of inducing further hepatic damage secondary to the interruption of pregnancy.

Opinion is divided as to which of these is the better course to follow. Both methods of management are good, and it will be a matter of individual difference in each patient as to which course should be followed. Should the latter course be followed and hepatic failure develop, management will be for hepatic failure as described previously. Delivery in this event should not be artificially induced but should be effected spontaneously. If hepatitis is too severe for the continuance of pregnancy, the patient will abort or deliver prematurely. Otherwise the patient will recover from the hepatitis and delivery can then be effected when the patient has sufficiently recovered. The problem thereby resolves itself.

In the delivery, general anesthetics are to be avoided as local infiltration is the anesthetic of choice. Uterine contractions are exceptionally painless in severe hepatitis, and little anesthesia is therefore required.[2] Prolonged bleeding is to be expected in the third stage of labor and during the first 24 to 48 hours postpartum. This bleeding may be adequately controlled by firm uterine contractions in the immediate puerperium; but later, when the uterus relaxes, delayed bleeding occurs because of a failure of the clotting mechanism in the uterine sinuses. Such bleeding is not amenable to vitamin K therapy, and Reid[47] believes the defect is due to decreased production of fibrinogen by the liver. The use of uterine tamponade or fibrinogen infusions may therefore be lifesaving.

In cases of mild hepatitis, it is still of value to give the patient therapeutic doses of vitamin K in the hope of preventing excessive blood loss. Blood loss in hepatitis is tolerated poorly and should be replaced immediately and completely. Adequate preparations should be made prior to the delivery for control of hemorrhage and immediate replacement of blood loss. Since the infectious agent is present in the mother's stools, special care must be exercised to prevent contamination of the newborn with the mother's

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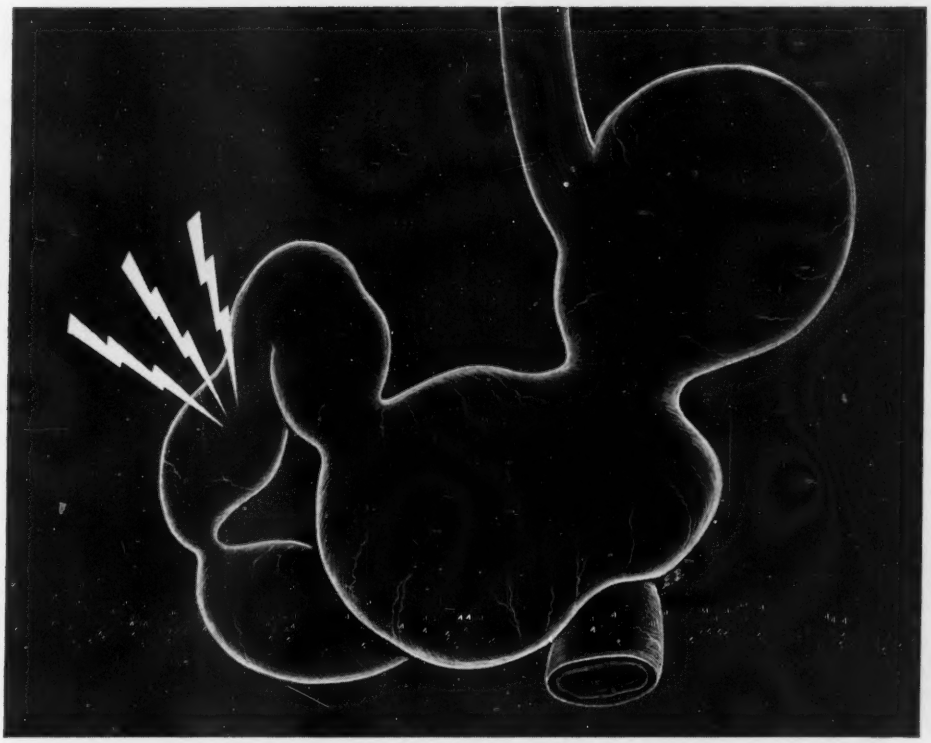

Abnormal Motility as the Cause of Ulcer Pain

Until recently the general opinion was held that ulcer pain was primarily caused by the presence of hydrochloric acid on the surface of the ulcer.

Present investigations[1,2] on the relationship of acidity and muscular activity to ulcer pain have led to the following concept of its etiologic factor:

> "... abnormal motility[2] is the fundamental mechanism through which ulcer pain is produced. For the production and perception of ulcer pain there must be, one, a stimulus, HCl or others less well understood; two, an intact motor nerve supply to the stomach and duodenum; three, altered gastro-duodenal motility; and four, an intact sensory pathway to the cerebral cortex."

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1. Schwartz, I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: A Clinical Evaluation of a New Anticholinergic Drug, Pro-Banthine, Gastroenterology *25*:416 (Nov.) 1953.

2. Ruffin, J. M.; Baylin, G. J.; Legerton, C. W., Jr., and Texter, E. C., Jr.: Mechanism of Pain in Peptic Ulcer, Gastroenterology *23*:252 (Feb.) 1953.

SEARLE

feces during the delivery. Management of the puerperium should receive added attention directed towards the hepatitis, following the principles of management previously mentioned.

What possible effect, if any, does maternal hepatitis have upon the developing embryo? A brief review of the known facts of teratology will help clarify the question. Developmental anomalies occur as a result of inherent germ plasm defects in the genes as inherited characteristics, or as a result of adverse conditions affecting the embryo. The latter consist of mechanical, actinic, endocrine, dietetic, and infectious factors.

Purely mechanical factors produce congenital defects such as occur in abdominal and tubal pregnancies and in abnormal uteri.[27,58] Radiation of maternal experimental animals and man has resulted in gross developmental defects of the newborn.[55, 58] Maternal diabetes is associated with increased fetal and neonatal mortality, macrosomia, and an increased incidence of congenital malformations.[52] That elevated blood sugar is not the definitive factor in the causation of these findings is illustrated by the fact that the prediabetic mother, whose blood sugar is normal at the time of delivery but who will later become diabetic, will exhibit the same incidence of fetal mortality, malformation, and macrosomia.[52]

Warkany[60] showed that dietary deficiencies of severe degree ended in termination of the pregnancy, and relative deficiencies were of importance in the production of abnormal offspring. Maternal rats maintained on vitamin A deficient diets gave birth to progeny with a fibrous rotrolenticular membrane replacing the vitreous. Riboflavin and vitamin D deficiencies produced specific skeletal defects in the progeny of maternal rats.[60] In man, beriberi is an example of maternal vitamin deficiency being transmitted to the infant.[52] Maternal infectious diseases exerting teratogenic effects upon the developing embryo were reported by Gregg[52, 62] in 1941, when he noted an unusually large number of congenital cataracts associated with congenital heart disease following an epidemic of maternal rubella in Australia.

These adverse factors that exert a teratogenic influence upon the fetus must exert their effect during the organogenetic period of gestation. The first eight weeks of gestation, when the velocity of cell reproduction is the greatest, is that period in which the embryo is most susceptible to cellular dysplasia.[56] It is possible that abnormalities may result as late as the 12th to the 14th week of gestation, although this less frequently occurs.

Stockard[59] demonstrated the importance of the time element of abnormal development. Using fish embryos, he demonstrated that different adverse conditions applied at the same phase of development tended to produce the same defects, whereas the same conditions applied at different phases of development produced different defects. Using case reports of maternal rubella, it was subsequently shown, by correlating the type of defect with the time of the maternal infection, that the critical period for developmental defects of the heart is the fourth to the ninth week, the lens the fifth to the eighth week, and the cochlea the seventh to the 12th week.[56]

Viral diseases other than rubella have been incriminated as exerting an effect upon the fetus in utero. Such an influence would occur as a result of placental permeability affording an avenue of infection.[27] Placental permeability is a consequence of: (1) the type of placenta concerned, a circumstance which varies with different species of animals; (2) the stage of pregnancy; (3) the size and weight of the substance in question; and (4) the selective activity which the placenta exerts in regard to certain substances.

Drugs such as penicillin, streptomycin, and morphine pass the placenta and can be demonstrated in the amniotic fluid and fetal blood. Vitamin C demonstrates the selectivity of the placenta, the concentration in the umbilical vein being several times greater than that of the maternal circulation.[52] Antitoxins, bacteriolysins, precipitins, and complement-fixing antibodies pass the placental barrier. Antibodies to influenza A, poliomyelitis, diphtheria, and mumps are transmitted from the mother to the child without significant decrease in titer.

Smallpox, variola, and varicella have been demonstrated as capable of infecting the fetus in utero.[52] There are recorded circumstances of infants born exhibiting characteristic lesions of the specific disease or developing the clinical picture of the disease within several days after birth. The period between birth and the clinical appearance of the disease is too short an interval of time to account for postpartum infection. Although these diseases can unquestionably infect the fetus in utero, resulting in stillbirth and abortion, there is no definite evidence available to prove any teratogenic effect upon the fetus.

Mumps, infectious mononucleosis, and herpes zoster are other diseases reported as possessing questionable teratogenic qualities. Case reports of fetal abnormality associated with these diseases have been recorded, but such cases are few in number, and no comparison is made of the incidence of the disease without fetal complications.

The question of whether or not the virus of infectious hepatitis possesses a teratogenic influence is likewise unsettled. There are case reports of fetal abnormalities associated with maternal infectious hepatitis. However, there is no proof that these abnormalities are a direct result of the virus infection and are not a result of chance coincidence associated with the infection. If the virus is to be incriminated as

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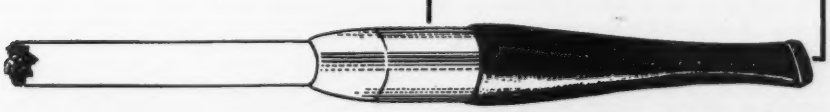

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having a teratogenic influence, such influence must be exerted during the first trimester. Otherwise, the principles of teratology are violated.

Of 40 cases of reported maternal hepatitis[6, 40, 50, 51, 53, 54, 57] and three cases at this center, in which the trimester of infection and the result of pregnancy are known, only two cases were complicated by congenital defects. In one case in which hepatitis occurred one month prior to and persisted throughout pregnancy, the result of gestation was a composite monster.[40] Infection occurred in the other case during the late second and early third trimesters and resulted in a hydrocephalic infant with microphthalmos.[54] There are three other cases in which hepatitis occurred in the first trimester.[50] One of these resulted in a normal term birth, and the other two resulted in spontaneous abortions.

These five cases illustrate several facts. Infectious hepatitis in the first trimester may result in spontaneous abortion, as shown by two cases. Fetal abnormality following hepatitis in the late second and early third trimesters cannot be accepted as the consequence of maternal hepatitis as this case does not satisfy the principles of teratology. This demonstrates chance coincidence of abnormality associated with but not a direct result of maternal hepatitis. Fetal abnormality in the one case of hepatitis complicating the first trimester may be the direct result of the virus of infectious hepatitis. This case satisfies the principles of teratology. The last case, a normal term birth following first trimester maternal hepatitis, demonstrates that even though such an influence may be exerted by the virus of infectious hepatitis, not every fetus will be so affected.

Since the question is unsettled, there is only one definite position that the physician may take regarding infectious hepatitis in the first trimester—that therapeutic abortion to prevent the birth of a congenitally malformed fetus is not to be practiced. Proof of a teratogenic influence is lacking. Even though such proof were available, normal fetuses would be sacrificed if therapeutic abortion were practiced, for not all cases of maternal hepatitis result in congenital malformations. Therefore, the question of management of the patient with first trimester hepatitis solves itself.

CONCLUSIONS

Infectious hepatitis is not a disease process peculiar to pregnancy. It is an infectious process complicating pregnancy, and interest is centered about the hepatitis.

Early diagnosis is difficult. Symptoms of the pre-icteric stage are misinterpreted as common phenomena of pregnancy. When icterus appears the diagnosis becomes easier. The differential diagnosis is the same as in the non-pregnant individual. Gamma globulin given prophylactically prior to the onset of symptoms prevents development of the disease.

Maternal and fetal prognosis is good when hepatitis is mild and grave when hepatitis is severe. Severe hepatitis occurs most frequently in the third trimester and progresses to acute yellow atrophy when treatment is not instituted promptly.

Treatment is directed toward hepatitis and consists of complete bed rest, adequate nutrition, and avoidance of hepatotoxic agents. Delivery should be accomplished by the simplest, easiest, and least disturbing method to maternal physiology.

Spontaneous abortion and premature delivery are frequent with hepatitis of moderate severity. A stillborn, macerated fetus is frequently encountered in acute yellow atrophy.

The question of a teratogenic influence exerted by the virus of infectious hepatitis is unsettled. Such an influence is possible, though sufficient evidence is not available to support a definite conclusion.

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THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL *reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.*

Cost of Medical Care

Human nature being what it is, people will complain about paying a comparatively modest sum for something they don't enjoy but must have—and will cheerfully spend much more for something they want, but don't urgently need.

The cost of medical care is a case in point. True, there are individual cases where serious trouble strikes a family and it seems hard-earned savings have to be spent because of prolonged sickness. However, at intervals there is bitter criticism of the bills charged by doctors and hospitals and pharmacists. An uninformed person might conclude that these costs generally are one of the biggest budget problems in the life of the average American.

A writer for the *Industrial News Review* says it isn't so and cites a brief pictorial chart in the June issue of *Medical Economics,* based on Department of Commerce estimates, to prove it. The figures show—

Each year we spend $1,600,000,000 for drugs—and $2,600,000,000 for items involved in personal care, such as cosmetics, lotions and toilet waters.

Each year we spend $2,600,000,000 in hospitals—and nearly twice as much, $5,300,000,000 for tobacco.

Each year we spend $2,800,000,000 for physicians' services—and well over three times as much, $8,800,000,000 for alcoholic drinks.

Each year we spend $2,900,000,000 for health care other than mentioned above—and over four times as much, $11,900,000,000 on recreation.

The fact that we have more fun smoking or drinking or playing and watching games than in visiting the doctor or the hospital should not be allowed to warp our attitude toward the facts.—*Emporia Gazette, July 13, 1955.*

Study of Free Medical Care

A survey conducted by the New Hampshire Medical Society recently and reported to the A.M.A. disclosed that some person in that state gets $4.00 worth of free medical care every minute. The society placed the annual value of this care at $2,096,640 a year, $40,320 a week, or $5,760 a day.

"Just about every practicing physician does some charity work," the report stated. "The scope depends upon the doctor's geographical location, the economic status of patients in his area, and the type of professional service rendered."

On the basis of its present active membership, the society credits the average doctor in the state with providing $3,425 worth of free medical care a year —slightly more than $65 weekly. For surgeons and other specialists the free care, measured in terms of dollars and cents, would be substantially greater.

New Edition of Infant Care

A new edition of *Infant Care,* the tenth revision since the publication for parents was first issued by the Children's Bureau, Social Security Administration, in 1914, came off the press last month.

A new section on the care of premature babies has been added, and increased emphasis is given to the need for precautions against accidents, the greatest single killer of children.

Like its predecessors, this edition attempts to bring together the best known and most widely accepted ideas of ways to keep babies physically healthy during their first year of life. A four-member Children's Bureau Pediatric Advisory Committee supervised publication.

The booklet is offered for sale by the Superintendent of Documents, Government Printing Office, Washington, D. C., at 15 cents a copy.

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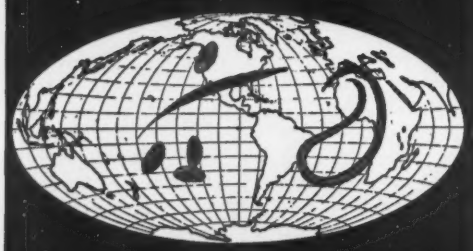

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Report of A.M.A. Delegate

Your delegates spent two and a half days in meetings during the annual session of the A.M.A. in Atlantic City, June 5-7, 1955. More than 80 resolutions and reports were received, referred to 13 reference committees, and finally acted upon by the entire House of Delegates.

DRUG STORE OWNERSHIP

The resolution from the Kansas delegates relating to this subject was thrown into the hopper, and the result was very satisfactory. It is, therefore, no longer unethical so long as the best interests of the patients are served and no proselyting is practiced.

HOSPITAL ACCREDITATION

The inflexibility of the standards set by the Joint Committee of Accreditation was fully discussed, there being several instances of what appeared to be resulting inequities. This simmered down to a few basic principles:

1. It is difficult to get trained examiners. Those who pass on the efficiency of the care of patients in various hospitals are physicians. These doctors also check staff organizations.
2. No hospital is accredited which has cultists caring for patients therein.
3. There is now a system of appeal whereby any hospital may show changes in proficiency entitling it to file for accreditation.

It is evident from the verbal report of the secretary of the Council on Medical Education and Hospitals that ignorance of the full operation of the system, on the one hand, and failure of many institutions to meet minimum standards, on the other, were the chief difficulties.

It became our opinion that the aggrieved institutions were as much or more at fault as the system of appraisal which doctors of medicine have developed.

It is true that the American Hospital Association appears at times to want to monopolize the program. We do not believe this to be either true or possible. It is still and should remain a cooperative effort in furnishing adequate care to our patients.

OSTEOPATHY

The so-called Cline Committee 25-page report had been received and studied by your delegates prior to the meeting. We of Kansas thought that it contained many ambiguities, generalities, and weaknesses, to the extent of being almost self-contradictory in describing osteopathic education in five of the six schools extant.

The delegation from Kansas was composed of Doctors Pyle, Eddy, Gsell, and Nelson, plus the valuable assistance of Mr. Rueben M. Dalbec, executive assistant of the Kansas Medical Society, and Mr.

H. Martin Baker, executive secretary of the Sedgwick County Medical Society. We held a council each morning at breakfast and outlined the day's activities such as delegation rooms to visit, luncheons to attend, and reference committee meetings which needed our attention.

Because of the directive given us by the House of Delegates of the Kansas Medical Society, we naturally concentrated on osteopathy. This was done with such intensity that objective reporting may be difficult.

The Cline report consisted of 25 typed pages prepared and signed by all the members of that committee and three medical school deans as advisors. The Kansas delegation individually and collectively studied this carefully. I believe the consensus of the group was that it contained many ambiguities, generalities, and self-contradictory statements. We selected the weaknesses which in general were these:

1. Throughout, the report indicated that none of the five osteopathic colleges met even the lowest minimum standards of medical education set up by our Council on Medical Education and Hospitals.

2. The osteopathic association has never publicly renounced the osteopathic (skeletal lesion) theory of disease or discontinued placing it in catalogues.

3. The osteopathic colleges and association have not officially asked the American Medical Association for help.

4. Practice rights problems belong to our several states and are not the concern of our national organization.

In various hospitality rooms, before the committee, and before the full House of Delegates, the Kansas delegation worked. The result was the acceptance of the committee minority report prepared by Dr. Milford O. Rouse of Dallas. I quote it verbatim:

MINORITY REPORT OF REFERENCE COMMITTEE ON MEDICAL EDUCATION AND HOSPITALS

Concerning report of Committee on Relations between Osteopathy and Medicine: One member of the Reference Committee was completely satisfied that an appreciable portion of current education in colleges of osteopathy definitely does constitute the teaching of "cultist" healing, and is an index that the "osteopathic concept" still persists in current osteopathic practice. Since he cannot with conscience approve the recommendation that doctors of medicine teach in osteopathic colleges where "cultism" is part of the curriculum, he respectfully makes the following recommendations to the House of Delegates:

1. That the report of the Committee for the Study of Relations between Osteopathy and Medicine be received and filed; and that the committee be thanked for its diligent work, and be discontinued.

2. That if and when the House of Delegates of the American Osteopathic Association, their official

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Bumbalo, T. S., Gustina, F. J., and Oleksiak, R. E.: J. Pediat. 44:386, 1954.

White, R. H. R., and Standen, O. D.: Brit. M. J. 2:755, 1953.

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Brown, H. W.: J. Pediat. 45:419, 1954.

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policy-making body, may voluntarily abandon the commonly so-called "osteopathic concept," with proper deletion of said "osteopathic concept" from catalogues of their colleges; and may approach the Trustees of the American Medical Association with a request for further discussion of the relations of Osteopathy and Medicine, the said Trustees shall appoint another special committee for such discussion.

Respectfully submitted,
Milford O. Rouse, M.D.

QUOTATION FROM OATH OF HIPPOCRATES

". . . and that by precept, lecture and every other mode of instruction I will impart a knowledge of the Art to my own sons and to those of my teachers, and to disciples bound by the stipulation and oath, ACCORDING TO THE LAW OF MEDICINE, BUT TO NONE OTHER."

To this delegate it seems a pity that we must spend so much effort negatively. There are positive efforts which could and probably should receive as much concerted effort: locally, the curtailment of other cultist healers who dispense such an inadequate brand of care to the unsuspecting public; nationally, ways and means to reduce the appalling automobile death rate; internationally, to further lower scientific medical barriers in the furtherance of world peace. These and many other positive efforts would seem more appropriate for the dignity and ethics of organized medicine.

Signed,
L. S. Nelson, M.D.
Senior Delegate from Kansas

Simplified Insurance Forms

Approval has been granted by the A.M.A.'s Council on Medical Service to five new simplified insurance claim forms drawn up by a special committee of the Health Insurance Council. The committee worked in collaboration with the Committee on Prepayment Medical and Hospital Service and included representation from all types of private insurance carriers. One simplified insurance claim form had previously been approved.

The additional claim forms may be identified by the following symbols and titles: ID-1, Attending Physician's Statement, Accident or Sickness (individual insurance); IDS-1, Attending Physician's Supplementary Statement (individual insurance); GD-1, Attending Physician's Statement (group insurance); GDS-1, Attending Physician's Supplementary Statement (group insurance); IPHS-1, Attending Physician's Statement, Accident or Sickness (individual hospital or surgical).

These five forms, together with GS-1 (group surgical expense, approved in 1954), are, in essence, adaptations of two basic forms—one designed for groups and the other for insurance underwritten on an individual or non-group basis.

The work has been done in the hope that insurance companies identified with the Health Insurance Council will use the forms in their day-to-day claims administration and that physicians throughout the country will co-operate by completing the simplified forms promptly to facilitate the administration of claims.

Three out of four traffic accidents involve passenger cars.

DEATH NOTICES

Robert Y. Jones, M.D.

Dr. R. Y. Jones, 73, an honorary member of the Reno County Society, died in Hutchinson on July 3 after an illness of two months. He had been in retirement since 1953. Dr. Jones was graduated from Rush Medical College in 1907, practiced two years in Chicago, and then opened an office in Hutchinson, specializing in surgery.

Wendell Maurice Tate, M.D.

Dr. W. M. Tate, 49, Peabody, died on July 12 in Axtell Hospital, Newton, after having suffered a heart attack two hours before. He was a member of the Marion County Medical Society. Dr. Tate was graduated from the University of Kansas School of Medicine in 1933 and served his internship at the Hertzler Clinic and Hospital in Halstead. He began practice in Peabody in 1935 and continued to practice there until his death.

Agnes Louise Robbins, M.D.

Dr. Agnes Robbins, 46, a member of the Wyandotte County Society, died at Bethany Hospital in Kansas City on July 15. She had been ill for some time. A graduate of the University of Kansas School of Medicine with the class of 1945, Dr. Robbins served her internship at St. Margaret's Hospital in Kansas City. She then opened offices in Kansas City and in Bethel. She was an active member of the American Academy of General Practice.

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THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

For more than a year the administration has been attempting to work out a system of voluntary, contributory health insurance for Uncle Sam's two million or so civilian employees and their families. It would seem a simple thing to arrange, considering that most big employers have had similar plans in operation for years. At any rate, the plan is ready now for Congress to act on, but putting it together hasn't been easy.

First, there was the question of how to fit in the many already existing health insurance plans (some conducted by U. S. employee unions), and at the same time to offer coverage to government people working and living where no adequate insurance is being offered.

Also, there was wide disagreement as to how much of the premium the federal government should pay; in private industry, employers' contributions range from a small percentage to the entire cost. U. S. employee unions naturally thought the federal government should set an example in generosity.

The program was first outlined early in the year. It then was put on the shelf for two reasons: a few refinements had to be made, and Congress first had to decide how big a pay raise it was going to allow U. S. workers this year before thinking about a fringe benefit, such as health insurance. The whole program was sent to the House and Senate just at the start of the adjournment rush, with the realization that not much could be hoped for this session.

The plan offers U. S. employees the option of signing up with a local non-profit service or indemnity plan, providing 75 per cent of the workers in the particular operation vote for a particular plan and providing that plan is approved by the U. S. Civil Service Commission. If the employees can't get together, or if no adequate plan is available locally, they can sign up for a uniform national indemnity plan to be underwritten by one or more large national insurance companies and negotiated by the Civil Service Commission. The proposed law itself lists specifically the original benefits that must be provided by the uniform plan, but authorizes the Commission to readjust them.

Regardless of which type coverage the employee selects for himself and his family, the federal contribution would be figured the same way. It could not exceed one-third of the total premium, or $19.50 annually for a single person or $52 for one with dependents, whichever figure is the lesser. If the uniform plan is chosen, the single employee could not be charged more than $39 annually, or the one with dependents more than $108 annually. But under any other plan, the employee would pay the difference between the U. S. contribution and the premium cost.

A system of major medical cost or catastrophic insurance also would be provided. Under it the employee would have to pay the first $100 of cost, after benefits of the basic policy had been exhausted, before major medical cost benefits would become available. From that point on, until $10,000 had been paid by the company, the employee would have to pay only 25 per cent.

The first major medical bill enacted was the extension for another two years of the doctor draft act, which for five years has been furnishing the armed forces and the Public Health Service with most of their doctors. Before passage, two changes were made in the law. The maximum age for induction was dropped five years. Under the old law a man could not be taken against his wishes after he had reached his 51st birthday; the new law reduced it to his 46th birthday. Also, the law no longer applies to physicians and dentists who have reached their 35th birthdays and who have been rejected for a medical or dental commission at any time solely on the grounds of physical condition.

Defense Department points out that the man has to be able to demonstrate that he actually applied for a medical or dental commission and was rejected; a 4-F draft board classification is not sufficient. The department also said that the law will not result in the discharge of men already in uniform, even though they could not be inducted under the new law.

As adjournment approached, prospects were that not much more medical legislation would be enacted this session. Most likely of success was a proposal for U. S. grants to states to help finance Salk vaccine costs; the states would decide the priority of age groups, but in a public program there could be no "means test" to determine whether a family could afford to pay. Under this plan the states would receive a certain amount as a straight grant, based on the state's economic need and the number of uninoculated children. If they wanted to put up dollar-for-dollar, the states also could draw on a second account. The bill does not set any limit on U. S. appropriations.

Two other possibilities were bills for a national survey of mental illness (which passed the House early in the session) and for U. S. grants to medical schools.

PHILEAS FOGG, MEET NELLIE BLY!

ENGINE 93 streaked through Arizona, its eight steel wheels flailing the track. And when the young lady at the controls thought the engineer wasn't looking, she opened up the throttle another notch.

She was Nellie Bly, reporter for the New York World. And she was in a big hurry to reach Jersey City and beat a fictional man in a trip around the globe. The man's name was Phileas Fogg, phlegmatic English hero of a popular novel by M. Jules Verne: *Around The World In 80 Days.*

And beat him she did—in just over 72 days—with only one dangerous incident. A "titled cad" tried to flirt with her in the middle of the Indian Ocean, but even he subsided when she threatened to signal the nearest U. S. man-of-war.

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ACTIVITIES OF MEMBERS

Dr. Ray T. Parmley, Wichita, is the author of a book, *Saddle Block Anesthesia,* published last month by Charles C Thomas, Springfield, Illinois.

Dr. Dennis Hardman, in the Navy for the past two years, has received his discharge and resumed his practice in Smith Center on July 1.

Dr. Clarence E. Thompson, Holyrood, is the sponsor of a "Sane Driving" contest for Holyrood youths between the ages of 15 and 21.

Dr. George J. Mastio, Jr., who has practiced in Ulysses for two years, closed his office there late in June and went to Kansas City to begin a residency in surgery at the University of Kansas Medical Center.

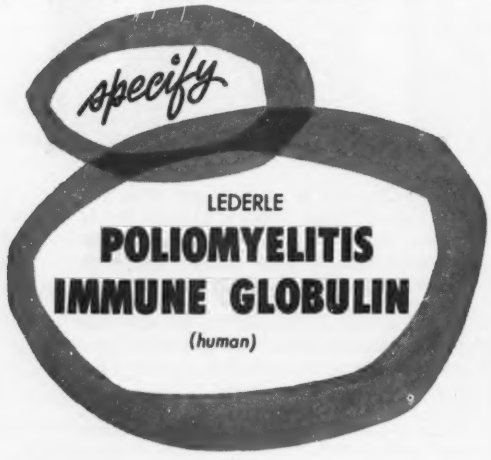
Dr. Hugh J. Woods, Smith Center, has gone to Kansas City for a year's postgraduate work in internal medicine at the University of Kansas Medical Center.

Dr. George L. Thorpe, Wichita, was elected vice-chairman of the Section on General Practice of the American Medical Association at the annual meeting in Atlantic City, June 11. Dr. Thorpe will become chairman of the section at the close of the annual meeting of the A.M.A. in Chicago, June 1956, and will preside at the meetings of the Section on General Practice and give the chairman's address at the A.M.A. meeting in Boston in June 1957.

Feature stories about Dr. J. W. McGuire, who has completed 50 years in practice, were published in June issues of the *Neodesha Register* and *Neodesha Daily Sun.* Dr. C. C. Dennie, Kansas City, Dr. James Butin, Chanute, and Dr. Lynn Beal, Fredonia, were among the speakers at an anniversary dinner in Dr. McGuire's honor.

Dr. Charles P. McCoy, Wichita, is taking a two-year postgraduate course in obstetrics and gynecology in St. Louis. He plans to return to Wichita later to practice in association with Dr. J. G. Kendrick and Dr. F. F. Nyberg.

Dr. George E. Burket and Dr. Sam Zweifel, Jr., Kingman, announce that Dr. Charles H. Finney, a 1954 graduate of the University of Kansas School of Medicine, is now associated with them in practice.



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Dr. Finney recently completed his internship at Sacred Heart Hospital, Eugene, Oregon.

Dr. Ernest C. Brandsted, McPherson, recently became a diplomate of the American Board of Obstetrics and Gynecology.

Dr. Robert L. Newman, of the University of Kansas Medical Center, spoke on "Ovarian Tumors" at a recent meeting of the Wichita Obstetrical and Gynecological Society.

Dr. Lawrence E. Filkin, who has been practicing in Concordia since 1946, became a member of the staff of the Gelvin-Haughey Clinic in Concordia recently. He specializes in obstetrics.

Dr. Paul M. Powell, Topeka, has announced that his son, Dr. Benson M. Powell, is now associated with him in practice. The younger Dr. Powell will limit his practice to surgery, specializing in thoracic surgery.

Dr. Clinton C. Fuller, Columbus, announces that Dr. Forrest Jones is now associated with him in practice. Dr. Jones, a graduate of the University of Kansas School of Medicine, recently completed his internship at St. Francis Hospital, Wichita.

Dr. Katherine Pennington recently completed a course in pediatric cardiology at Johns Hopkins Hospital, Baltimore, and has returned to her practice in Wichita.

Dr. Karl Menninger, Topeka, and Dr. W. Clarke Wescoe, dean of the University of Kansas School of Medicine, are now serving as members of a committee appointed by Governor Fred Hall to advise on matters affecting penal and correctional institutions in Kansas.

Three physicians were recently appointed by Governor Fred Hall to four-year terms on the Advisory Commission for the Norton Tuberculosis Sanatorium, Dr. H. Preston Palmer of Scott City, Dr. J. Lloyd Morgan of Emporia, and Dr. Robert M. Brooker of Topeka.

The El Dorado Clinic announces that Dr. Ben E. White is now a member of its staff. Dr. White, a graduate of the University of Kansas School of Medi-

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cine, recently completed internship at St. Margaret's Hospital, Kansas City.

Dr. Franklin D. Murphy, chancellor of the University of Kansas, delivered the commencement address at graduation exercises at the University of Pennsylvania Medical School in June.

Dr. LaRue W. Owen, who recently completed a residency in anesthesiology at Harvard Medical School, has returned to Wichita and is now practicing there.

Dr. Lee S. Fent, who recently completed a tour of duty with the Army at Fort Leavenworth, has returned to his practice in Newton.

Dr. William E. Mowery, a surgeon, and Dr. Robert D. Lindeman, who will specialize in obstetrics, have joined the staff of the Mowery Clinic in Salina. The clinic was founded by Dr. Mowery's father, the late Dr. W. E. Mowery.

The Bethel Clinic, Newton, announces that Dr. Francis N. Lohrenz, an internist, is now a member of its staff. Dr. Lohrenz was graduated from Vanderbilt University Medical School in 1949 and completed his training, internship and residency, at the University of Kansas Medical Center.

A feature story about Dr. Albert N. Gray, who recently celebrated his 85th birthday, was published in the *Burlington Daily Republican* on July 1. Dr. Gray has practiced for 47 years and has begun his 38th year in the Burlington community.

Dr. Arnold Greenhouse, a graduate of the University of Kansas School of Medicine, has joined the staff of the Dodge City Medical Center. Dr. Greenhouse has been a resident in internal medicine at the University of Kansas Medical Center during the past three years.

Announcement has been made of the appointment of the following new health officers: Dr. Melvin H. Waldorf, Jr., Greensburg, for Kiowa County; Dr. Carroll D. Behrhorst, Winfield, and Dr. Roscoe F. Morton, Arkansas City, for Cowley County, and Dr. J. D. Smith, Satanta, for Haskell County.

Dr. Albert W. Schmidt, who has been limiting his practice in Lyons to two hours a day in recent years,

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has announced his full retirement. He plans to spend the remainder of the summer in Colorado.

The Chamber of Commerce in Lincoln is making plans for a ceremony to be held in September to honor Dr. L. A. Kerr, who recently retired after 50 years of service to the Lincoln community.

Dr. Harlan W. Berthelsen, who formerly practiced in Sedan, is now located in McPherson. He is occupying officers formerly maintained by Dr. Robert P. Watterson, who recently moved to Arizona.

Dr. M. H. Waldorf, Jr., Greensburg, announces that Dr. Robert Fenton is now associated with the Bradley-Waldorf clinic there. Dr. Fenton is a graduate of the University of Kansas School of Medicine.

Dr. John F. Montague, who has been practicing in St. Marys, closed his office on August 1 to begin a residency in orthopedic surgery at Franklin Hospital, San Francisco.

The Nelson Clinic, Manhattan, announces the addition of Dr. Leonard A. Wall to its staff. Dr. Wall was graduated from the University of Oklahoma Medical School in 1951 and served his internship at Wesley Hospital, Oklahoma City. Since 1952 he has been a resident in obstetrics and gynecology at the University of Kansas Medical Center.

Film on Medical Education

"Danger at the Source" is the title of a new 13½-minute film produced recently by the National Fund for Medical Education. It tells the story of medical education in America, prepared as a public service by Fox Movietone and narrated by Ray Middleton.

Filmed in medical schools and teaching hospitals and supervised by medical educators, the picture highlights the glories and hard work of the American system of medical teaching. It is designed to emphasize the importance of the nation's 81 medical schools and the problems they face in accumulating the necessary funds to continue as a free and progressive force for the national welfare.

The National Fund for Medical Education, founded in 1949, has distributed nearly $7 million in unrestricted grants to schools. The goal is $10 million each year. The address of the fund is 2 West 46th Street, New York 36, New York.

The 1956 annual meeting of the Kansas Medical Society will be held in Topeka, April 29-May 3.

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BOOK REVIEWS

Obstetrics. Eleventh Edition. By J. P. Greenhill, M.D. Published by W. B. Saunders Company, Philadelphia. 1008 pages, 1170 illustrations. Price $14.

The new edition of Obstetrics by Greenhill (formerly Dee Lee and Greenhill) is a complete revision of the old text which was out of date in many respects. The new book contains complete discussions of practically all facets of obstetrics and is well documented with references to the recent literature. The illustrations are excellent, and the type is easy to read. The style is lucid; discussions are detailed and specific enough to make the book a handy reference manual. It is at present the best general obstetrics textbook available.—*E.W.J.P.*

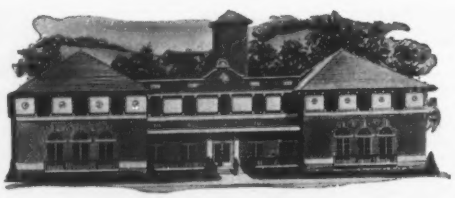
Pomp and Pestilence: Infectious Disease, Its Origins and Conquest. By Ronald Hare, M.D. Published by Philosophical Library, Inc., New York. 224 pages. Price $5.75.

This excellent, readable little book should be read by everyone interested in the broader aspects of bacteriology. The author, after a brief but interesting account of the origin of man and of parasites, discusses some of the best known pathogenic species and describes their life cycle, modes of infection, and the diseases they produce.

In Chapter Two the author considers the place of origin of many well-known infectious diseases and the methods by which they were scattered from their original focus. Chapter Three contains a most interesting history of smallpox, plague, typhus, cholera, and influenza. The author points out the role great epidemics have played in the history of nations, pointing out that, while the Romans were fighting the Germans on the borders of the empire, an epidemic of smallpox in Rome was playing havoc with the Roman population.

The chapter on plague is of extreme interest. The author points out again the impact of the plague of Justinian on history. He points out that so-called bacteriological warfare is not so modern since the Tartars in their war with the Genoese in the 14th century hurled into the city of Caffa the bodies of patients who had died of the plague.

The role of typhus is also portrayed most vividly. The author describes in considerable detail the epidemic of typhus fever in Ireland—the mass evictions by absentee landlords and large scale emigration. "There can be little doubt that typhus had much to



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do with the Anglophobia which still affects many of the citizens of the U. S. A."

Later chapters in this work describe the effectiveness of many public health measures later introduced. In the closing chapter, after discussing the inadequate food supply for the teeming millions in China, India, Indonesia, and Burma, the author makes the statement that, with improved sanitation and health measures, these people may be able to save their children from death from parasitic infection but be unable to save them from death from starvation. "The prospect for the future is therefore bleak in the extreme. But whatever is done or not done, parasites will be taking a hand in the proceedings. . . ."

This interesting little book should be read to be appreciated and re-read to be fully appreciated.—*R.H.M.*

Radioisotopes Policy of AEC

The Atomic Energy Commission has made radioisotopes for all biomedical and agricultural research and research in medical therapy available to domestic users at 20 per cent of catalogue price, effective on July 1, 1955. Reduced prices heretofore available only for cancer research will be extended for other projects.

The plan was broadened for three reasons: (1) radioisotopes have proven useful in study of other important human diseases; (2) the distinction between cancer research and fundamental studies in biology, biochemistry, and biophysics is not always clear; (3) stimulation of the use of radioisotopes in the life sciences is considered to be in the public interest.

Under the new policy, radioisotope users will make application to the Atomic Energy Commission, Division of Biology and Medicine. Investigators or institutions awarded the discount must agree to publish results of their research.

Public Relations Institute

The 1955 Public Relations Institute sponsored by the A.M.A. will be held at the Drake Hotel, Chicago, August 31 and September 1. Medical society officers and personnel, public relations committee chairmen, and members of the Woman's Auxiliary to the A.M.A. are invited to attend.

Among topics to be considered are: grass roots activity in national legislation, basic public relations techniques, medical publicity in magazines, and the individual's role in communication. There will also be opportunities for exchange of ideas, discussion of future A.M.A. activities, and viewing of motion pictures.

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ANNOUNCEMENTS

The 1955 convention of the National Society for Crippled Children and Adults will be held at the Palmer House, Chicago, November 28-30.

The 33rd annual scientific and clinical session of the American Congress of Physical Medicine and Rehabilitation will be held August 28-September 2 at the Hotel Statler, Detroit. All sessions will be open to physicians in good standing with the A.M.A. Full information may be obtained from the secretary, 30 North Michigan Avenue, Chicago 2, Illinois.

The Southwestern Surgical Congress announces a change in dates for its seventh annual meeting, to be held in Kansas City, Missouri, at the Hotel Muehlebach. The new dates, one week earlier than those previously announced, are Monday, Tuesday, and Wednesday, September 5-7.

Dr. William P. Longmire, Los Angeles, Dr. Frederick L. Reichert, San Francisco, and Dr. J. Englebert Dunphy, Boston, will be guest speakers and will serve as moderators at panel discussions. Each day's program will also include a feature somewhat like a cine clinic, one presented by St. Louis University School of Medicine, one by the University of Kansas School of Medicine, and the third by Washington University School of Medicine.

Non-members are invited to attend and may secure programs by writing Central Office, 207 Plaza Court Building, Oklahoma City 3, Oklahoma.

The American Urological Association offers an annual award of $1,000 for essays on the result of some clinical or laboratory research in urology. First prize will be $500, second prize $300, and third prize $200. Competition is limited to urologists who

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Historical Material Needed

In preparation for the observance of The Kansas Medical Society's centennial anniversary, members of the Committee on History are attempting to collect all material of historical interest. Physicians who can contribute information, records, etc., are urged to send such to

Committee on History
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have been graduated no more than 10 years and to men in training to become urologists.

Information may be secured from the secretary, 1120 North Charles Street, Baltimore. Essays must be in his hands before December 1, 1955.

Creighton University School of Medicine, together with the Nebraska State League for Nursing and the Nebraska Arthritis and Rheumatism Foundation, will sponsor a three-day institute on rehabilitation of neuromuscular diseases and rheumatic diseases at Creighton Memorial St. Joseph's Hospital, September 8-10. Programs may be secured from Harold N. Neu, M.D., Department of Rehabilitation, Creighton Memorial St. Joseph's Hospital, Omaha.

Postdoctoral fellowships ranging from $4,000 to $6,000 per year, tenable for one year with prospect of renewal, are being offered by the Arthritis and Rheumatism Foundation, 23 West 45th Street, New York 36, New York. Senior fellowships for more experienced investigators will carry awards of from $6,000 to $7,500 per year and are tenable for five years.

The 62nd annual convention of the Association of Military Surgeons of the United States will be held at Hotel Statler, Washington, D. C., November 7-9, 1955. The association's offices are at Suite 718, New Medical Building, 1726 Eye Street, N.W., Washington.

The Council on Postgraduate Medical Education of the American College of Chest Physicians, in cooperation with its respective state chapters and the staffs and faculties of hospitals and medical schools of Chicago and New York, will sponsor two fall postgraduate courses on diseases of the chest. The first will be at Hotel Knickerbocker, Chicago, October 3-7, and the second will be at Park-Sheraton Hotel, New York, November 14-18. Tuition for each is $75. Complete information may be secured from Executive Director, 112 East Chestnut Street, Chicago 11, Illinois.

The Eighth Annual Mid-West Cancer Conference, sponsored jointly by the Kansas Division of the American Cancer Society and the Committee on Control of Cancer, Kansas Medical Society, will be held at the Broadview Hotel, Wichita, March 22 and 23, 1956.

"Endocrinology and Metabolism" is the subject of the seventh annual postgraduate assembly of the

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Endocrine Society, to be held in Indianapolis, September 26-October 1, at Indiana University Medical Center. Information may be secured from Postgraduate Office, 1100 West Michigan, Indianapolis 7, Indiana.

Importance of Small Hospitals

The hospital with 100 beds or less is the backbone of our nation's hospital care, reports Mr. Ray E. Brown, president-elect of the American Hospital Association. He bases that statement on the fact that of 5,212 general and short term hospitals, 3,533 are small institutions. They admit 5.5 million patients a year and record 836,500 births annually.

"Small hospitals face many problems in their efforts to fulfill their tremendous responsibility to the communities they serve," he said. "Even the smallest hospital today has to be a complete medical unit." He reported that even those of the smallest size must maintain most of the services found in large hospitals to keep pace with research and modern technology.

Records of the American Academy of General Practice show that 31 Kansas physicians attended the Academy's 1955 scientific assembly in Los Angeles.

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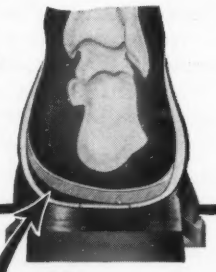

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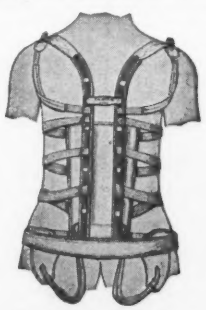

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Since 1945 Congress has appropriated 10 million dollars to research in the field of rehabilitating amputees. A number of physicians, scientists, engineers, and prosthetists have devoted full time to the problem, and many schools are carrying on research programs of their own. The Department of Physical Medicine at the Buffalo school has already pioneered in the field by setting up research projects for amputee types considered unfit by others, the old, the very young, and those with multiple disabilities.

Heart disease is the "most formidable mountain" in medical research, in the opinion of Graydon L. Walker, vice-president of Parke, Davis and Company, who spoke to a group of druggists recently on important medical problems now to be solved. Heart disease, he reported, "is the Mount Everest on the graph which shows the range of mortality statistics.

"Each time we reach the summit in a successful fight to conquer a fatal disease, the patients who are saved become candidates in later years for heart disease, cancer, nutritional disorders, or one of the remaining uncontrolled infections—the mountains on our horizon."

History shows that tuberculosis is the product of incomplete civilization. It is a struggle in which civilized man is still at a biological disadvantage because imperfectly adapted to the new mode of life which he is creating or which is forced upon him. Tuberculosis is a social problem involving many factors other than the tubercle bacillus. It will be rapidly mastered only if society can be more perfectly fitted to human needs, limitations, and urges.—*Rene J. Dubos, Ph.D., NTA Transactions, May, 1954.*

Good health cannot be forced upon the public. We can, however, create an environment in which people will study their health needs and work out ways of doing what they want to do with what they have. This is true health education and the essence of democracy.—*Leroy E. Burney, M.D., Am. J. Pub. Health, Feb., 1955.*

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Hot Weather Nutritional Tests

One hundred volunteer airmen began a two weeks' field test on July 6 to aid in the development of an improved global survival ration, according to a release from Headquarters Air Research and Development Command. The men subsisted on diets ranging from starvation to normal.

Half the men exercised heavily and the other half lightly. Further breakdowns were made on controlled amounts of drinking water and unlimited amounts to determine the effects of water on the airmen's well being. In all, 20 different feeding situations were studied, varying in caloric content and nutrient mixture in terms of fats, proteins, and carbohydrates. Physiological studies of the men were made for two weeks before the start of the test and were continued for two weeks afterward.

Basically, the 100 men were divided into four major groups.

Group I was permitted unlimited amounts of water, light exercise, and a predetermined amount of food, ranging from no food for some up to 3,000 calories for others.

Group II received the same rations of food and water as Group I, but the men were required to exercise heavily.

Group III received similar food rations, exercised lightly, and received only limited amounts of water.

Group IV received food and water rations similar to Group III but exercised heavily.

The tests were conducted by the University of Illinois at Camp Atterbury, Indiana, with volunteers living in the field.

A cold weather field test was conducted last winter at Camp McCoy, Wisconsin. It demonstrated that a crashed airman's best chances for survival in cold climates depend on liberal quantities of drinking water and a ration made up of approximately 15 per cent protein, 52 per cent carbohydrate, and 33 per cent fat.

The decline in infectious disease, first of acute and more lately, and quite as conspicuously, of chronic illness, has led to longer life, larger populations, and a change in the main type of disease to which the existing population is subject. The additional persons and extra years mean at the same time greater needs for sustenance and more productivity in meeting them. Somewhere, unless the earth's resources are augmented in presently unknown ways in better provision of space and expanded food supply, an equalization must be attained in population additions and subtractions.—*Esmond R. Long, M.D., Bull. of the Hist. of Med., July-Aug., 1954.*

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NO ONE IS COMPLETELY IMMUNE

BONAMINE* HCl

BRAND OF MECLIZINE HYDROCHLORIDE

Motion sickness affects people of all ages because almost everyone is sensitive to labyrinthine irritation induced by travel on land and sea and in the air.

Bonamine has proved unusually effective to prevent and treat this minor but distressing complaint. And a new agreeable method of administration is now offered by the incorporation of this well-tolerated agent, with its prolonged action, in a pleasantly mint-flav[illegible] drug co[illegible] minutes

Bonami[illegible] nausea, [illegible] labyrint[illegible] Menière

Supplied:

Bonamine Tablets *(scored and tasteless) 25 mg.*

New
Bonamine Chewing Tablets *25 mg.*

DATE DUE

*TRADEMARK

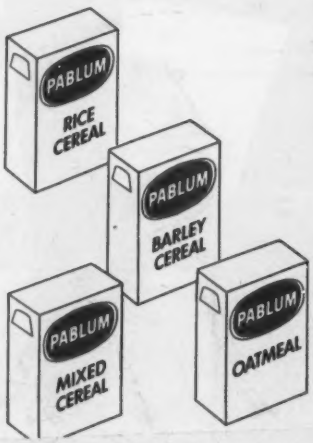
Pfizer **PFIZER LABORATORIES,** Brooklyn 6, N.Y.
Division, Chas. Pfizer & Co., Inc.

Why so many physicians SPECIFY PABLUM CEREALS

TOMMY started on Pablum Rice Cereal at the age of 2 months. He likes its smooth texture (all Pablum Cereals are smooth). Pablum Cereals give him plenty of iron—½ oz. supplies 4.2 mg.—to help prevent iron deficiency anemia.

MARY LOU likes Pablum Oatmeal. Since she has been eating Pablum Cereals her growing appetite is satisfied longer.

Pablum Rice Cereal
Pablum Barley Cereal
Pablum Oatmeal
Pablum Mixed Cereal



BARBARA—like other children—enjoys *all* four Pablum® Cereals. Each variety tempts her awakening taste buds. Pablum Cereals are scientifically packaged to insure freshness. The 'Handi-Pour' spout is an extra convenience for busy mothers.

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